THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY.

Vol. VIII. JANUARY, 1928. No. 31.

Original Papers.

THE PROGNOSIS AND THE LATE RESULTS OF POSTENCEPHALITIC RESPIRATORY DISORDERS.*

By W. ALDREN TURNER and MACDONALD CRITCHLEY, London.

INTRODUCTION.

The disorders of respiration associated with epidemic encephalitis have been the occasion for a considerable literature, mostly of a descriptive nature. Efforts have also been made to indicate a pathogenesis, although the problem cannot be said to have emerged from the stage of hypothesis. There has hitherto been no effort to trace the end-results of patients suffering from these respiratory disorders, and consequently no certain prognosis has been applicable.

In a previous work on the respiratory disorders in encephalitis Turner and Critchley[14] modified the original classification of Marie and Lévy[12], and referred to (1) disorders of respiratory rate, (2) disorders of respiratory rhythm, and (3) respiratory tics. The first group included cases of tachypnœa and of bradypnœa; in the second category the cases of breath-holding spells, apnœic pauses, and Cheyne-Stokes respiration were placed. The third group comprised such disorders as spasmodic cough, involuntary yawning, blowing, sniffing, etc. Dealing with the problem of prognosis, the authors state: "two main questions naturally arise . . . first, is the appearance of respiratory symptoms of itself a feature of serious import? And, secondly, how long do the respiratory disorders persist? . . . There is a certain amount of evidence to suggest that the appearance of tachypnœa . . . is of grave outlook in the older patients, particularly when associated with pyrexia . . . The duration of these symptoms seems in the majority of cases to be indefinite, although now and again a case will improve spontaneously. . . . The majority of cases however tend to persist with unabated violence for an indefinite length of time. They show no tendency to progress, however; this prognosis apparently holds good for all the varieties of respiratory disorders."

Such was the position in the spring of 1925; since that date other cases have come under observation, and many more isolated case-reports have been published. Some of these latter have been recorded as "cured" by some

* Work carried out under the auspices of the Medical Research Council.

particular therapeutic measure, such as freezing of the phrenics (Gamble *et al*[5]), by psychoanalysis (Witzel[16], Jelliffe[8,9,10]) or by hypnosis (Runge[13]). The object of this paper is purely utilitarian: its aim has been to determine if possible the ultimate fate of patients showing this particular variety of sequela. The cases originally reported by us have been followed up and to these have been added others which have come under our personal observation or been reported to us through the kindness of other physicians.

We have not included in this paper acute cases in which respiratory manifestations were present. Hall's[6] conclusions with regards to these patients will be referred to.

LITERATURE.

The literature of encephalitis, despite its magnitude, contains surprisingly little reference to the prognosis of the respiratory syndromes. A few isolated reports can however be traced in which cases of this variety of sequela have been followed up over some length of time.

The first instance of postencephalitic respiration anomaly quoted in the United States was reported by Aronson[1] (1921). His patient, a boy of 8, showed tachypnœa and dysrhythmia: the author propounds the query as to the ultimate fate of this child: although the case is quoted by Turner and Critchley as having cleared up in six months, such is apparently not the fact, for in a personal communication to Jelliffe made in 1926, Aronson stated that there was no recovery, and that the boy had developed "some interesting regressive character anomalies."

In 1922 Mlle. Lévy[11] reported in her monograph *Les manifestations tardives de l'encéphalite* numerous examples of respiration disorder. Case 35 of her series was that of a woman of 35 who developed tachypnœa and Parkinsonism after acute encephalitis in 1920. After 30 months there was an apparently perfect recovery. Another patient (Case 37), a male of 29, developed a "puffing" respiratory tic, which was still present two years later. Case 62 concerned a boy of 10 who developed blowing and sniffing tics which came on in paroxysms. Each attack brought about such exhaustion that he dropped off to sleep. His conduct deteriorated so much that he could no longer be retained at the Salpêtrière but was transferred to St. Anne and later to the Vanclure. His respiratory tic was still present two years later. Her last patient (Case 67), a girl of 14, was admitted to the Salpêtrière nine months after her acute attack, with Parkinsonism and a tic-like cough. Polyuria and polydipsia were also present. Her respiratory tic still continued at the time of publication, viz., nine months afterwards.

Runge[13] in 1923 gave a very full report of a case of postencephalitic Parkinsonism with tachypnœa and tetany arising in a young student. The case was investigated from a psychological standpoint and inherent psychoneurotic trends were revealed. Psychoanalysis exposed a masturbatory compulsion and under hypnotic treatment over a period of one year the patient definitely improved.

In the same year, Fiamberti[4] reported the case of a girl of 12 who after encephalitis developed Parkinsonism and paroxysmal tachypnœa. At the onset of each attack the child would bend forwards nearly double and then abruptly rise erect, clutch her throat and give a forced expiration. The sphincters frequently became relaxed. Attack would follow attack until she ultimately fell into a sleep of exhaustion. Eleven months after coming under observation she fell ill with paratyphoid fever; during her convalescence the paroxysms of disordered breathing abruptly left her and did not return.

At the 1923 Conference of the British Medical Association Hinds Howell[7] mentioned the case of a female patient, age 20, who developed dyspnœa after encephalitis. This symptom after persisting for three weeks improved but later relapsed. At the time of announcement the respiration had become normal, though the length of time after the original illness was not stated.

Wimmer's monograph on Chronic Encephalitis[15] (1924) contains an account of a girl, age 18, who showed attacks of yawning. Gradually these spells left her, only to be replaced by paroxysms of tachypnœa, severer in nature than the original disability.

Witzel[16] (1925) records the histories of two patients: the first one, a girl of 14, developed character changes and rapid laboured breathing after encephalitis. Under psychotherapy the respiration became normal. His other patient (Case 4), a boy of 13, was subject to spasms of noisy panting respirations. There was no history of acute encephalitis, and no mention is made of the presence of any abnormal physical signs. This patient also became well after psychotherapy.

In the same year, Gamble, Pepper and Miller[5] gave details of a patient of 18, who developed a severe tachypnœa one year after an attack of encephalitis. There were other evidences of central nervous disease, including a right-sided hemianæsthesia, weakness of the face, nystagmus, and sluggish pupillary reactions to light. With a view to checking the tachypnœa, both phrenic nerves were exposed in the neck and blocked by freezing with ethyl chloride. During this operation the respiration rate fell from 96 to 20 per minute. No further tachypnœa occurred up to the time of publication in October, 1925 (*i.e.*, nearly seven months).

A case of paroxysmal breath-holding reported in 1925 by Hall, Mayers and Kerr[17], concerned a youth of 19 who developed encephalitis in 1920. He first came under treatment for his respiratory disorder in 1923; there was no Parkinsonism. He left hospital on July 8th, 1923, but returned the following February with a well-marked adiposogenital syndrome. During this interval the attacks of breath-holding had entirely ceased. The respiration remained normal up to the time of publication.

One of the latest additions to the literature is contained in the Medical Research Council's report on the 1924 epidemic of encephalitis in Sheffield (issued in 1926). In their report Hall and Gurney Yates[6] investigated the prognostic significance of various of the sequelæ; they dealt *inter alia* with

respiratory manifestations and considered them separately, according as they arose during the acute phase or as an after-effect. Of the former group of respiratory disorders they stated: "There is a distinct suggestion that its presence in the primary attack is of somewhat serious significance as regards life. Thus only 33 per cent. have recovered, 28 per cent. have not recovered and 39 per cent. died. . . . This mortality is more than double that in the cases as a whole, and exceeds considerably that of any other single symptom amongst those investigated. . . . Of the 18 cases in which the breathing was disturbed in the acute attack, not one showed any similar or other respiratory abnormality at the end of 1925." Regarding the second group of cases (numbering 20 in their series) they state: "Late or residual disorders of the respiratory mechanism are not usually preceded by anything similar in the primary attack. They are not of such serious importance as regards life." The fate of the patients *qua* respiratory disorder is not dealt with.

Jelliffe [8,9,10] reported on several occasions an account of a postencephalitic respiratory disorder occurring in a male of 21; polydipsia, polyuria, character changes and Parkinsonism were present in addition to spells of tachypnœa and breath-holding. In some of these attacks, tetany would develop. Under psychoanalysis—carried out over a period of six to seven months—the paroxysms ceased and remained absent for a period of 12 to 18 months (*i.e.*, up to time of publication in January, 1927). We were informed by Jelliffe in October, 1927, that the breathing has remained normal and that some improvement is noticeable in the degree of Parkinsonism.

In the Annual Report (1926-1927) of the Metropolitan Asylums Board Borthwick [2] publishes interesting details of some cases of respiratory disorder treated in the Encephalitis Unit of the Northern Hospital, Winchmore Hill. Amongst these is the case of a girl of 14 who suffered from frequent paroxysms of breath-holding; during the period of inspiratory apnœa the diaphragm descended to an extremely low level, and the heart became grossly distorted. There were no physical signs of Parkinsonism. The child—whose acute attack had occurred in July, 1924—had been a patient since February, 1926; when observed in October, 1927, *i.e.*, 20 months after her admission, the respiratory attacks were still present with unabated violence and frequency.

PERSONAL CASES.

The material upon which the present analysis is based comprises primarily the original seven cases reported in our first paper. Unfortunately one case in this series (No. V) has been lost sight of. In addition we have dealt with 23 other cases, most of which have occurred in the practice of the National Hospital, Queen Square. For several of the cases we are indebted to the courtesy of Professor Hall, of Sheffield; through the kindness of Dr. Feiling, Dr. C. P. Symonds, Dr. Pearson and Dr. Greig Anderson we are also able to include those of some other patients.

We have considered the outcome of these cases under five main heads, viz. :—

A. Cases of complete cure.
B. Cases in which respiration has become normal although the mental or psychical concomitants are unchanged or worse.
C. Cases in which respiration has improved, although there has been no corresponding alteration in the associated manifestations.
D. Cases in which respiratory disorders persist unchanged.
E. Fatal cases in which death occurs during the phase of respiratory disorder.

A. *Cases of apparent cure.*

We have only met with two cases in our experience which can be regarded as a cure, in that all respiratory symptoms have disappeared, and there have been no mental or physical sequelæ characteristic of encephalitis in their place. Our first case was actually one of the severest cases of paroxysmal breath-holding which we have ever encountered, and was reported in full in our original communication.

1. H. B., male, age 16 (patient of Dr. Turner, at Queen Square), had a mild attack of acute encephalitis in April, 1924. He came into hospital in the following September because of frequent attacks of breath-holding, accompanied by cyanosis, mental obfuscation, and many bizarre grimaces and contortions. There were no character changes and no physical signs of Parkinsonism. He remained in hospital six months, during which time no marked change occurred either in the frequency or character of the respiratory disorder. A week after discharge he was sent to the Berkshire Mental Hospital, having developed auditory hallucinations. He believed he heard the devil whispering to him and telling him to hold his breath.

He was seen by one of us at the asylum in November, 1925. He had lost a good deal of weight ; his breathing was noisy, rapid and laboured, but the breath-holding paroxysms were less severe and less frequent. He complained of pain in the right shoulder and inability to raise the arm above a right angle. The right hand was wasted, but there were no objective sensory changes.

Christmas, 1926, while in the asylum, the patient developed a severe attack of influenza. On recovery his breathing was almost normal and he since has gradually recovered. He rapidly regained weight, and his delusions and hallucinations left him entirely.

He was seen again by one of us in May 1927, when his breathing was perfectly normal : he looked well, and discussed his previous mental illness candidly and freely. He was still unable, however, to raise the right arm above a right angle. There was no trace of Parkinsonism.

In August 1927 (*i.e.*, exactly three years after the onset of respiratory symptoms), he was again seen. He was still weak in the right arm ; there was no wasting, but there was an area of analgesia embracing the whole of the right upper arm, the right shoulder, and the right side of neck, and thorax, back and front. There was a sharp line of demarcation at the edge of the chin and at the elbow joint. The condition was obviously a hysterical one and under gas the shoulder joint could be freely manipulated in all directions.

The completeness of the recovery in this instance was almost dramatic. His paroxysms during the active stage were of such severity and frequency as to suggest an outlook of the gravest possible type. He was becoming progressively weaker and rapidly losing weight. The disappearance of respiratory

symptoms during a severe febrile illness recalls the similar case of Fiamberti, in which the breathing became normal after an attack of paratyphoid fever. A feature of extreme interest lay in the appearance of a paralysis of the arm; although probably arising in the first place as the result of trauma, such as might be occasioned by the excessive contraction of the accessory muscles of respiration, the condition of the arm when last seen was clearly one of hysteria. There was no limitation of passive movements under anæsthesia, and in addition there was a wide area of analgesia which was sharply delineated and corresponded with no known neural segmentation. The patient has no retrograde amnesia; he can discuss without emotion the details of his hallücinosis.

2. H. L. H., male, age 16, came under the care of Professor Hall in November 1924. His acute attack had occurred in the previous February. When seen in November he had no evidences of objective change in his nervous system, but complained of frequently recurring "blowing" attacks, accompanied by nausea and vertigo. In the severer attacks tetany supervened, and though there was no actual loss of consciousness some degree of mental confusion occurred.

His physical state in February 1927 (two years after the onset) had not changed, but the dysrhythmia had ceased. For the past year he has been in continuous daily employment, and has been attending in addition a night school three evenings weekly.

B. *Cases in which respiratory disorder has ceased but other physical or mental sequelæ have appeared or become worse.*

3. V. W., female, age 15, was admitted to Queen Square under the care of Dr. Birley in November 1924. There was a history of an acute attack of encephalitis in the previous April. Whilst in hospital she exhibited an inverted sleep rhythm, slight twitching of the corner of the mouth, and involuntary protrusion of the tongue. There was spasmodic torticollis and a very mild degree of left-sided Parkinsonism. At night time her respiration became noisy, rapid and irregular, being interrupted by periods of apnœa.

She left hospital in January 1925, but her mother could not keep her at home because of a gradually increasing change in disposition. She "turned against her little sister," threw a chair at her mother, and threatened suicide.

In January 1926, she was admitted to West Park Asylum, Epsom. She was there visited by one of us at the end of July 1927 (two years and eight months after first coming under observation). She then showed a moderately severe grade of Parkinsonism, especially on the left side. There were no longer any involuntary movements of the face, tongue or neck. Mentally she was childish, and very quarrelsome. She was retarded and becoming progressively slower in cerebration. She exhibited an abnormal craving for attention and sympathy, even to the length of inflicting bruises and cuts upon herself.

No respiratory disorder whatever was complained of by her or noted by the attendants, either at night time or by day.

4. E.S., male, age 19 (case of Dr. Collier, at Queen Square), first came under treatment in January 1925. His acute attack dated from March 1924. His physical state was one of mild Parkinsonism with rhythmic myoclonus in the deltoids, trapezii, and pectorals of both sides. Breathing was rapid (40 per minute) and noisy.

He left the wards of the National Hospital but continued to attend the out-patient department regularly.

When seen in August 1927 (two years and seven months afterwards), his Parkinsonism had enormously advanced, so that he was completely helpless. He was unable to make the effort of speaking. His respiratory disorder had disappeared six months previously and there were no traces of myoclonus in the shoulder muscles.

This case was reported on one or two occasions, and pneumographic records were made to show a synchrony between the rate of the myoclonic jerks and the respiration.

5. F.M., male, age 24 came into hospital in April, 1925 (Dr. Kinnier Wilson, Queen Square). An acute attack of encephalitis had occurred exactly one year before. At the time of his admission he presented the picture of Parkinsonism of moderate severity. For the past two months his breathing had become a little noisy and difficult. His chief respiratory anomaly consisted in attacks of bradypnœa in which the respiratory rate fell to 12 per minute. Thoracic expansion was of good amplitude.

In June 1927 (*i.e.*, two years and two months after first coming under observation) all traces of disordered respiration had gone. His Parkinsonism syndrome was still present and there was in fact an aggravation in some of the symptoms, especially the salivation.

6. B.A., female, age 14, first came under observation in January 1926 (Dr. Birley, at Queen Square). Her acute attack had occurred between four and five years previously. At the time of admission she complained of spasmodic, involuntary jerking movements of the head in a backward direction. This movement was accompanied by a wide opening of the mouth and a sharp, noisy inspiration. The head then slowly returned to its normal position and at the same time the mouth closed and the breath was expelled slowly and quietly. The movement occurred at two-minute intervals when she was conscious of being watched, but was less frequent when her attention was distracted. There were no gross physical signs in the nervous system beyond irregularity of the pupils and slight right facial weakness.

The chief change noted in July 1927 (18 months later) was a very definite dysarthria. The head movements and inspiratory tic had ceased.

7. W.G., female, age 17, was first seen (Prof. A. J. Hall) in September 1925. She had suffered her acute attack in February, 1924. When first seen she was definitely Parkinsonian and had periodic attacks of noisy tachypnœa. These had been present for nine months. The attacks were very frequent and lasted from three minutes to half an hour. They were accompanied by tetany, cyanosis and mental confusion; the eyes were often fixed in an attitude of extreme left lateral deviation. A period of acapnia usually supervened after each paroxysm.

She was seen again just two years afterwards; she had not had a single attack for the past year. On the other hand, her conduct had become much altered; she had developed into an incorrigible liar and thief; her Parkinsonism had not advanced.

8. J.W., male, age 16 (case of Prof. A. J. Hall) first came into hospital in March 1924, for insomnia and paroxysms of noisy tachypnœa, accompanied by tetany. The acute phase dated from 12 months previously. His physical condition was one of Parkinsonism of mild degree.

His physical state in September 1927 (three years and six months later) had not become worse, but he had now developed oculogyric crises; his conduct had also deteriorated and he had become subject to maniacal attacks with violence. The respiratory disorder had entirely disappeared.

9. L.K., female, age 9 (case of Prof. A. J. Hall), had her acute attack in March 1924, and came into hospital in December 1924, with Parkinsonism and frequent attacks of noisy tachypnœa. Her physical state in August 1927 (two years and eight months later) had become worse and she was now an inmate of a mental hospital suffering from intense depression. Her breathing attacks had left her some months ago.

10. E. W., male, age 20 (case of Prof. A. J. Hall), had his acute attack in March 1924, and three months later began to develop mild attacks of rapid breathing. In September 1925, signs of Parkinsonism were noticeable in his left arm. In September 1927 (*i.e.*, two years and three months after the onset of tachypnœa) his Parkinsonian syndrome was worse, but the attacks of rapid respiration have ceased.

11. R.W., male, age 17 (case of Prof. A. J. Hall). The acute attack dated from March 1924; In October of the same year attacks of gasping first appeared.

Six months later (by March 1925) his respiration became normal. His physical state in November 1925, was one of Parkinsonism; no further respiratory anomaly has appeared.

12. A.M., male, age 9, was admitted to the National Hospital (Dr. Kinnier Wilson) on account of nocturnal restlessness. His acute illness had occurred about 19 months previously (*i.e.*, in the Spring of 1924). Whilst in hospital his day-time conduct was exemplary, but towards nightfall he became noisy, excited, and turbulent. He would run about the ward blowing hard through his fists as though he were playing a musical instrument, and it was not until the small hours of the morning that he would drop off into a sleep of exhaustion. There were no signs of Parkinsonism. He was transferred in February 1926, to the Northern Hospital Encephalitis Unit, Winchmore Hill. Here his behaviour steadily grew worse; his blowing tic persisted up to February 1927, when it gradually left him. When seen in October, 1927 (*i.e.*, 23 months after his admission to the National Hospital) there were still no signs of Parkinsonism; his respiratory disorder had ceased; his conduct however was certainly worse than it was two years previously.

C. *Cases in which respiratory disorder has improved though not disappeared.*

Within this group we place a few cases which in the course of time have shown some improvement in the breathing attacks, either as regards frequency or severity or both. The physical concomitants may, as in both the following cases, simultaneously get worse.

13. W.E., male, age 55, first came under observation in 1924 (Dr. Phillips, Southmead Hospital, Bristol). He had been treated for acute encephalitis six weeks previously. When at Southmead Hospital there was a moderate degree of Parkinsonism, and rhythmic myoclonic movements of the left shoulder muscles. He was subject to paroxysmal attacks of noisy tachypnœa of very shallow amplitude "like the panting of a dog which has been running."

He was often in and out of hospital in the next three years. When visited in July 1927, by one of us (two years and seven months after first being seen), he was in a state of very severe Parkinsonism. He made no complaint of breathlessness or thoracic distress, but was troubled with intense vertigo. The amplitude of the chest during ordinary slight breathing was fair, but perhaps a little less than normal. When told to take a deep breath in and out, his respiration became somewhat rapid (28 per min.).

His attacks of tachypnœa still occurred though much less frequently and with less severity. They were particularly prone to come on when in bed at night.

14. E.B., male, age 31 (in-patient at Queen Square, under Dr. Howell), was admitted in January 1926. His acute attack of encephalitis dated from 1924. His respiratory disorder, which had been present five months, consisted in attacks of noisy tachypnœa in which he would stand up first of all with his trunk bent forwards to a right angle, breathing noisily and rapidly, and then suddenly straighten up and bend backwards in an opisthotonic attitude. He exhibited a mild degree of Parkinsonism.

In June 1927 (*i.e.*, 18 months after first being observed), his attacks were still present, but less severe in nature and less frequent in occurrence. He has now developed tremor in addition to his general attitude of Parkinsonism.

D. *Cases in which there has been little or no change in the severity of the respiratory disorder.*

15. H.W., male, age 26, admitted to Queen Square (Dr. Kinnier Wilson), in December 1924.

The acute attack occurred in January 1920, and was followed six months later by the gradual onset of Parkinsonism. At the time of his admission the P.A. syndrome had

reached a moderate degree of severity. He was, moreover, subject to incessant attacks of involuntary yawning in which his mouth would gape to a surprising extent. No other respiratory disorder was present, and there were no psychical changes.

His state in June 1927 (two years and seven months later), had shown no material change. The yawning bouts were as severe and as frequent as ever. The Parkinsonian state had not advanced to any appreciable extent.

16. E.F., male, age 52, was admitted in July 1926, under Dr. Collier (Queen Square). He had suffered his attack of encephalitis in December 1924. There was no trace of Parkinsonism and no psychical changes. His respiratory anomaly consisted in grotesque attacks of breath-holding, in which he stood up and took several deep, noisy breaths in rapid succession. Next he stiffened himself and slowly bent the trunk backwards, at the same time extending his arms and abducting them at the shoulder joints. The forearms and arms alternately flexed and extended. There was sometimes a transient loss of consciousness during these attacks, in which the patient would fall.

Twelve months later his paroxysms were still continuing with unchanged severity and frequency, and no fresh symptoms had appeared.

A short note on this patient appeared in a previous paper[3] but was not included in the original series.

17. S.T., male, age 8, was admitted into Queen Square (Dr. Birley) in April 1924, with an acute attack of encephalitis with choreiform movements. He left hospital in the middle of May, apparently cured.

Soon after returning home he became noisy and restless at nights and developed attacks of noisy and rapid breathing, aggravated by excitement or by fatigue.

When examined in July 1927, (two years and three months later) there was no evidence of Parkinsonism, but the bouts of noisy tachypnœa were still present.

18. Male, age 14, first came under observation (Dr. A. Greig Anderson) in July 1924. His original illness had apparently occurred in June 1924. On July 1st, 1924, he was extremely restless and sleepless; no objective signs were discovered in the central nervous system except an ill-sustained nystagmus. In November of the same year he developed attacks of noisy tachypnœa, still not sleeping well; examination revealed a doubtful plantar response on the right, and nystagmus on right lateral ocular deviation. In June 1927 (*i.e.*, two years and seven months later) he was still having attacks of tachypnœa.

19. E.W., male, age 24, attended the out-patient department of Queen Square (Dr. Birley) in May 1926, for involuntary gaping movements of the lower jaw, which had been present for two months. His acute attack had occurred in June 1924; there was a condition of Parkinsonism of moderate severity. He attended the out-patient department regularly and up to the date of communication.

In September 1927 (*i.e.*, one year and four months later) there was no material change either in his Parkinsonism or in his gaping.

20. W.D., male, age 33, attended the out-patient department of Queen Square (Dr. Howell), suffering from Parkinsonism, palilalia, oculogyric crises and paroxysms of breath-holding, ushered in by a very long and audible nasal inspiration. His attack of acute encephalitis dated from January 1924. The respiratory trouble had been present for two and a half years and still continued to date (June 1927).

21. M.H., female, age 11, attends the out-patient department at Queen Square (Dr. Adie). Her encephalitis occurred in 1924; her present state is one of Parkinsonism and character changes. She worries other children, torments and assaults her sisters. For the past three years she has been subject to bouts of tachypnœa with a loud expiratory phase. There has been no change in the severity or frequency of these attacks during the last three years.

22. R.G., male, age 14, a patient of Queen Square (Dr. Holmes), whose acute attack dated from May 1924, is now in a condition of Parkinsonism. In the spring of 1925 he became addicted to a respiratory tic in which he would give a series of loud and abrupt

expirations through his nostrils. He was admitted to hospital in May 1927, and improved considerably; the tic relapsed however in July and is still present in all its former severity.

23. K.D., female, age 12, was admitted to Queen Square (Dr. Kinnier Wilson) in June 1926. She had suffered from encephalitis some time in 1924, and her present complaints were of dyspnœa and audible tachypnœa. There was a mild degree of Parkinsonism present.

In September 1927 (*i.e.*, one year and three months after leaving hospital) her physical condition had improved; except for her expressionless face there was no trace of Parkinsonism. The mother stated that she had become wilful of late and had developed a habit of stopping strangers in the street and engaging them in conversation. The respiratory disorder was still present and had not noticeably altered in character. One new feature, however, has arisen; many of her breathing attacks now end in sleep; she drops her head, and shuts her eyes for about two minutes; she is very easily aroused from this sleep.

The occurrence of these narcoleptic-like attacks following the paroxysms of tachypnœa is of especial interest. Sleep has been reported by both Mlle. Lévy and Fiamberti, coming on at the end of bouts of disordered breathing. In their patients, however, the sleep appeared to be one of exhaustion, whereas in our case, there was no preceding fatigue or background of sleepiness. The unconsciousness would come on suddenly; it was irresistible; it resembled normal sleep in character and was promptly terminated by extraneous stimuli.

24. F.H., female, age 27, attended the out-patient department of Guy's Hospital (Dr. Symonds) with a mild Parkinsonian syndrome. Her acute attack dated from December 1925. Since May 1926, she had been subject to a paroxysmal dry, hacking cough, coming on towards evening. There was no adequate cause for the cough discoverable in the chest or larynx. At times she also complained of breathlessness causing her to take every now and then a respiration of deeper amplitude. When seen in September 1927 (15 months later), the respiration disorder was still present.

25. A.H., male, age 30, an out-patient of Guy's Hospital (Dr. Symonds), sustained an attack of encephalitis some time in 1924. A year later he began to complain of numerous indefinite symptoms resembling those of neurasthenia and in addition "fits" of a vague nature made their appearance. In January 1927, he began to suffer from attacks of tachypnœa and sighing respiration particularly at meal times.

In September 1927 (nine months later), the respiratory disorder was still present, and there was no trace of Parkinsonism.

26. P.A., male, age 17, an out-patient of Queen Square (Dr. Howell), dated his original attack of encephalitis from March 1925. He slowly developed a paralysis agitans syndrome of moderate severity, and six months before coming under observation he began to have attacks of involuntary gaping. These paroxysms persisted with variable intensity, but there was no real change in their severity or frequency over a course of six months.

27. A.B., male, age 19, was admitted to the National Hospital (Dr. Holmes) for Parkinsonism. His acute attack dated from some time in 1925. For one year prior to admission his breathing had been subject to paroxysms of noisy acceleration. In September 1927 (*i.e.*, 16 months after the onset of the tachypnœa) his rapid breathing still persisted with its usual severity.

E. *Fatal Cases.*

Death may supervene both in cases of acute encephalitis with respiratory manifestations or—less commonly—in cases of chronic or subacute encephalitis in which breathing anomalies have coexisted.

28. L.B., male, age 5, was admitted to Charing Cross Hospital in January 1925, (Dr. Pearson). The acute illness had occurred in April 1924, and was followed by restlessness, insomnia, and choreiform movements. When first admitted the child was

awkward on its legs; could not talk intelligibly; it was very drowsy by day and would sometimes fall asleep over its meals. At night-time the child was intensely restless. The expression was vacant, the mouth gaping widely.

Three months later the head became retracted and Kernig's sign was present. The spinal fluid was normal. The child's respiration now became characteristically changed. It was irregular and there were periods of apnœa followed by a spell of noisy exaggerated breathing with marked expiratory phase. During the apnœic period there were simultaneous involuntary movements of the lower jaw and the left hand.

Four months after admission the child's condition had markedly deteriorated. It had become cachectic, and could swallow only with difficulty. The limbs and trunk were still more hypertonic; irregular twitching movements of the left hand were visible. The temperature rose to 103° at night. There was no change in the character of the respiration. This state lasted a month and the child eventually succumbed.

29. A.C., male, age 25 (patient of Dr. Feiling, Hospital for Epilepsy and Paralysis, Maida Vale) sustained his acute attack of encephalitis in March 1924. He thereafter developed a condition of Parkinsonism, together with paroxysms of tachypnœa with a cogwheel expiratory phase. The Parkinsonian state gradually increased and in April 1927 (*i.e.*, 37 months after the onset of the acute illness) he died. Dr. Hunt Cooke kindly wrote to us as follows: . . ." the irregular and peculiar respiratory condition persisted until he became comatose about a week before he died. He then had Cheyne-Stokes breathing."

Case No. 29 was among the seven cases recorded in our original paper. In this patient fluoroscopic examination of his diaphragm showed that the rise during expiration consisted of a series of rapid jerky movements. This 'cogwheel phenomenon' was also demonstrable in the pneumographic tracings.

We have purposely omitted from this section any record of fatal acute cases in which the terminal phase was characterized by acceleration of the respiration rate. A consideration of such does not come within the scope of this present paper.

DISCUSSION.

In all, some 29 cases have been followed up over periods extending to three years: the results have been variable and may be set out thus:—

A.—Cured	2
B.—Respiratory disorder cured but other manifestations worse or unchanged	10
C.—Respiratory disorder improved but other manifestations worse or unchanged	2
D.—Respiratory disorder unchanged	13
E.—Death	2

One patient could not be traced.

It appears therefore that the respiratory disorder disappeared in 12 out of 29 cases and improved in two others: in the remaining 15 cases it has persisted unchanged up to time of observation and in two instances till death.

(a) *The variety of respiratory disorders.*

In Table 1 the results are set out according to the variety of the original breathing disorder. Tachypnœa was the commonest variety, comprising 15 cases. Of these, nine were cured (as far as the respiration was concerned) when followed up over periods extending from 27 to 42 months: one patient

expirations through his nostrils. He was admitted to hospital in May 1927, and improved considerably; the tic relapsed however in July and is still present in all its former severity.

23. K.D., female, age 12, was admitted to Queen Square (Dr. Kinnier Wilson) in June 1926. She had suffered from encephalitis some time in 1924, and her present complaints were of dyspnœa and audible tachypnœa. There was a mild degree of Parkinsonism present.

In September 1927 (*i.e.*, one year and three months after leaving hospital) her physical condition had improved; except for her expressionless face there was no trace of Parkinsonism. The mother stated that she had become wilful of late and had developed a habit of stopping strangers in the street and engaging them in conversation. The respiratory disorder was still present and had not noticeably altered in character. One new feature, however, has arisen; many of her breathing attacks now end in sleep; she drops her head, and shuts her eyes for about two minutes; she is very easily aroused from this sleep.

The occurrence of these narcoleptic-like attacks following the paroxysms of tachypnœa is of especial interest. Sleep has been reported by both Mlle. Lévy and Fiamberti, coming on at the end of bouts of disordered breathing. In their patients, however, the sleep appeared to be one of exhaustion, whereas in our case, there was no preceding fatigue or background of sleepiness. The unconsciousness would come on suddenly: it was irresistible: it resembled normal sleep in character and was promptly terminated by extraneous stimuli.

24. F.H., female, age 27, attended the out-patient department of Guy's Hospital (Dr. Symonds) with a mild Parkinsonian syndrome. Her acute attack dated from December 1925. Since May 1926, she had been subject to a paroxysmal dry, hacking cough, coming on towards evening. There was no adequate cause for the cough discoverable in the chest or larynx. At times she also complained of breathlessness causing her to take every now and then a respiration of deeper amplitude. When seen in September 1927 (15 months later), the respiration disorder was still present.

25. A.H., male, age 30, an out-patient of Guy's Hospital (Dr. Symonds), sustained an attack of encephalitis some time in 1924. A year later he began to complain of numerous indefinite symptoms resembling those of neurasthenia and in addition "fits" of a vague nature made their appearance. In January 1927, he began to suffer from attacks of tachypnœa and sighing respiration particularly at meal times.

In September 1927 (nine months later), the respiratory disorder was still present, and there was no trace of Parkinsonism.

26. P.A., male, age 17, an out-patient of Queen Square (Dr. Howell), dated his original attack of encephalitis from March 1925. He slowly developed a paralysis agitans syndrome of moderate severity, and six months before coming under observation he began to have attacks of involuntary gaping. These paroxysms persisted with variable intensity, but there was no real change in their severity or frequency over a course of six months.

27. A.B., male, age 19, was admitted to the National Hospital (Dr. Holmes) for Parkinsonism. His acute attack dated from some time in 1925. For one year prior to admission his breathing had been subject to paroxysms of noisy acceleration. In September 1927 (*i.e.*, 16 months after the onset of the tachypnœa) his rapid breathing still persisted with its usual severity.

E. *Fatal Cases.*

Death may supervene both in cases of acute encephalitis with respiratory manifestations or—less commonly—in cases of chronic or subacute encephalitis in which breathing anomalies have coexisted.

28. L.B., male, age 5, was admitted to Charing Cross Hospital in January 1925. (Dr. Pearson). The acute illness had occurred in April 1924, and was followed by restlessness, insomnia, and choreiform movements. When first admitted the child was

awkward on its legs; could not talk intelligibly; it was very drowsy by day and would sometimes fall asleep over its meals. At night-time the child was intensely restless. The expression was vacant, the mouth gaping widely.

Three months later the head became retracted and Kernig's sign was present. The spinal fluid was normal. The child's respiration now became characteristically changed. It was irregular and there were periods of apnœa followed by a spell of noisy exaggerated breathing with marked expiratory phase. During the apnœic period there were simultaneous involuntary movements of the lower jaw and the left hand.

Four months after admission the child's condition had markedly deteriorated. It had become cachectic, and could swallow only with difficulty. The limbs and trunk were still more hypertonic; irregular twitching movements of the left hand were visible. The temperature rose to 103° at night. There was no change in the character of the respiration. This state lasted a month and the child eventually succumbed.

29. A.C., male, age 25 (patient of Dr. Feiling, Hospital for Epilepsy and Paralysis, Maida Vale) sustained his acute attack of encephalitis in March 1924. He thereafter developed a condition of Parkinsonism, together with paroxysms of tachypnœa with a cogwheel expiratory phase. The Parkinsonian state gradually increased and in April 1927 (*i.e.*, 37 months after the onset of the acute illness) he died. Dr. Hunt Cooke kindly wrote to us as follows: . . ." the irregular and peculiar respiratory condition persisted until he became comatose about a week before he died. He then had Cheyne-Stokes breathing."

Case No. 29 was among the seven cases recorded in our original paper. In this patient fluoroscopic examination of his diaphragm showed that the rise during expiration consisted of a series of rapid jerky movements. This 'cogwheel phenomenon' was also demonstrable in the pneumographic tracings.

We have purposely omitted from this section any record of fatal acute cases in which the terminal phase was characterized by acceleration of the respiration rate. A consideration of such does not come within the scope of this present paper.

DISCUSSION.

In all, some 29 cases have been followed up over periods extending to three years: the results have been variable and may be set out thus:—

A.—Cured	2
B.—Respiratory disorder cured but other manifestations worse or unchanged	10
C.—Respiratory disorder improved but other manifestations worse or unchanged	2
D.—Respiratory disorder unchanged	13
E.—Death	2

One patient could not be traced.

It appears therefore that the respiratory disorder disappeared in 12 out of 29 cases and improved in two others: in the remaining 15 cases it has persisted unchanged up to time of observation and in two instances till death.

(a) *The variety of respiratory disorders.*

In Table 1 the results are set out according to the variety of the original breathing disorder. Tachypnœa was the commonest variety, comprising 15 cases. Of these, nine were cured (as far as the respiration was concerned) when followed up over periods extending from 27 to 42 months: one patient

had died. The remaining six cases remained unchanged when seen at a period of nine months to three years later. Spells of breath-holding constituted the next commonest type, five cases belonging to this group: of these patients, one was cured, one had improved, and one died: the other two had improved. Of the three cases of paroxysmal yawning, all remained *in statu quo* after intervals from six months to over two and a half years. The other cases of tics (coughing, blowing, etc.), numbered four in all, two of which remained as before and two improved. One patient only was observed with bradypnœa. In this case the respiration became normal although the Parkinsonism increased.

(b) *Influence of the Parkinsonian Syndrome.*

The effect of an associated state of Parkinsonism is shown in Table II. In 20 cases, the patient was also afflicted with some degree of the paralysis agitans syndrome. Of these, one patient died. In eight, the respiratory disorder was unaltered: in two it improved and in nine it disappeared. The fate of the breathing anomaly bore no parallelism with the severity of the syndrome or with its course.

The nine cases in which no Parkinsonism coexisted included one fatal case, three cures, and five in which the respiration did not change.

(c) *The presence of psychotic manifestations.*

The possible influence of concomitant disorders of conduct or character upon the prognosis of respiratory disorder offers a suggestive avenue of enquiry, and may have a bearing upon the causation of the abnormal breathing. Psychotic changes of a gross character were present in eight only of this series: of these, one became well, and in five the respiration became normal although the other manifestations did not improve. In the other two instances the respiration remained unchanged (see Table III).

Of the remaining 21 patients in whom there was no obvious behaviour or character change, two died, six became well, two improved, and eleven remained *in statu quo.*

(d) *The age factor.*

Fifteen of our patients were in the second decade of life: three were in the first, and six in the third: two patients were in the fourth and fifth respectively and one only in the sixth. Males outnumbered females in our series, the proportion being twenty-one to eight.

(e) *The date of the original attack.*

Although the literature shows that respiratory disorders may follow encephalitis whichever epidemic the original illness dates from, it is the experience of most in this country that the 1924 epidemic was responsible for the majority of the cases of postencephalitic respiratory disorders which are seen to-day. This impression is supported in our series: Table IV indicates that no less than 23 out of 29 cases date the original illness from the 1924 epidemic.

Reference to official figures, however, indicates that a proclivity of the 1924 cases to develop respiratory disorders is apparent rather than real, for the epidemic of that year was the severest known in this country. The number of notifications is almost double those occurring in the following year, which marked the next most severe epidemic.

THE VARIABILITY OF THE RESPIRATORY DISORDERS.

Postencephalitic respiratory disorders are in most instances paroxysmal in appearance, and it is of value to consider those factors which appear to influence the severity or frequency of the attacks. In the majority of cases no precipitating cause can be traced: their proximity is heralded at times by prodromata of thoracic, laryngeal or nasal discomfort, and the disordered breathing follows. Frequently it is noticeable that the attacks occur only towards nightfall, and in such there seems to be a close association with fatigue states: the converse condition, however, in which the subject is at his worst in the early hours of the morning, shortly after waking, is not uncommon. In a few patients the respiratory disorder invariably follows the awakening from sleep, irrespective of the hour. Occasionally (Case 3) the respiratory anomaly occurs only during sleep, and the patient is ignorant of any abnormality.

Emotional stress is perhaps the most potent aggravating factor in these cases: almost all these patients are at their worst when the object of scrutiny, or when under the influence of fright, annoyance, or embarrassment. Pleasurable excitement, however, may be accompanied by a respite from the attacks, as in Cases 14, 16, and 23. Boredom or lack of interest in the environment is usually associated with an aggravation: thus we are told by the wife of patient No. 14: "He is better when occupied: he can go out for a five mile walk or a motor ride and not have an attack: but the trip must be one of interest to him: he must *want* to go out." If on the other hand he is reluctantly led out by his wife for a stroll the attacks are frequent. During concentration, or occupations entailing mental or physical effort, there may be a diminution in the paroxysms: thus, one patient (Case 14) was fond of music and would like to compose and play pianoforte music for an hour at a stretch, during which time he would be free. The act of shaving however was always attended by unpleasant associations and he was invariably worse whilst so occupied.

The effect of intercurrent illness or noxious stimuli in diverting the respiratory disorder is noteworthy. In Case 1, for example the patient was almost free from attacks whilst suffering from pneumonia. We are informed that another patient (Case 14) was once taken out for a walk against his will; instead of the attacks being worse as was anticipated his breathing was normal because "he had very bad chilblains on his feet and these hurt him intensely as he walked." A temporary freedom coinciding with a feverish cold has been noted in more than one case.

The act of eating has been known to exert an unfavourable influence in a few cases: a patient (Case 15) tells us: "It all depends upon the amount of

phlegm I have in my mouth; when I have a lot my mouth opens. . . . It is always worse when I am eating or when my food is lying heavy on my chest."

Severe physical exertion as a rule has little or no influence upon the appearance of respiratory disorders: alterations of posture are occasionally possible factors in curtailing or launching an attack.

The ability to cut short an attack is rarely present to any marked extent on the part of the patient himself. At times we hear that by exerting a very determined effort of will the respiratory disorder may be checked, but usually for a while only. The effort of self-control apparently entails so much distress that the patient abandons it and finds it easier to endure the paroxysm. Extraneous stimuli are more potent; by speaking sharply to the patient, or by distracting the attention, the breathing disorder may sometimes be checked. More often it is possible to correct the respiration by directing the patient's attention towards his own performance and by ordering him to hold his breath or to take deep, slow inspirations. The effect however is rarely more than temporary, and becomes less efficacious on repetition.

In no case in our experience has any permanent benefit resulted from such measures as heavy dosage with sedatives, from electrotherapy, or from the inhalation of oxygen, nitrous oxide, or carbon dioxide. Of the cases in the present series which cleared up, no association could be demonstrated with any particular therapeutic measure: the majority were actually on no treatment at all.

There is an obvious similarity therefore between the results of precipitating and inhibiting factors and the phenomenology of hysteria, but the association must not be strained. The power of temporary inhibition of disordered breathing at the instigation of external suggestions argues merely that the volitional or corticobulbar controlling mechanism is intact; the aggravation of the attacks during emotional stress and during introspection illustrates a phenomenon occurring in every type of organic disease in which consciousness is intact.

It seems therefore that though diurnal variability is the rule—dependent upon the play of internal and external factors—the influence of such stimuli is but slight in degree and transitory.

Occasionally the respiratory disorder changes in type; thus a particular patient may show at the onset of his illness a paroxysmal cough which later is replaced by paroxysms of tachypnœa, or of breath holding. Such was the case with Nos. 1 and 23 in our series (cf. also Wimmer's case 12).

SUMMARY.

Although the number of cases which form the basis of this paper is too small to justify definitive conclusions as to prognosis, it is possible that these cases constitute a representative example of postencephalitic respiratory disorders in general. In the 29 cases investigated, almost every variety of known breathing anomaly is represented; in general terms it may be said that nearly

one half of the cases have either ceased to show any respiratory disorders, or else have shown them to an attenuated extent. An equal proportion of cases remained unchanged over a period of time, extending up to three years. Two patients have died, whilst still giving evidence of a respiratory disorder.

It would seem that the character of the breathing anomaly does not materially alter the prognosis, although the small series of cases investigated will not allow of any conclusive statement. The proportion of recovery cases appears to be greater in the cases with a progressive Parkinsonian state than in those without the manifestation of paralysis agitans.

Although it is indubitable that numerous physical and psychical stimuli may temporarily influence the severity of the disorder, there is no evidence that these factors are other than temporary.

It must be emphasized that the recovery rate apparently does not depend upon any particular treatment; improvement when it occurs seems to appear spontaneously.

We beg to thank the Physicians of the National Hospital for permission to quote many of the cases in this series.

REFERENCES.

[1] ARONSON, L. S., "Encephalitis with unusual sequelæ," *Neur. Bull.*, 1921, iii, 113.

[2] BORTHWICK, G. A., "Some Observations on the After-effects of Epidemic Encephalitis in Children," *M.A.B. Annual Report*, 1926-27, 165.

[3] CRITCHLEY, M., "The Pathogenesis of Respiratory Anomalies after Epidemic Encephalitis," *Brit. Med. Jour.*, 1927, ii.

[4] FIAMBERTI, A. M., "Encefalite epidemici cronica, a tipo respiratorio con esito in quarigione," *Note e Riv. di Psychiat.*, 1923, vi, 363.

[5] GAMBLE, C. F., PEPPER, O. H. P. and MILLER, J.P., "Postencephalitic Tic of the Diaphragm. Pulmonary Over-ventilation and Relief by Blockage of the Phrenic Nerves," *Jour. Amer. Med. Assoc.*, 1925, lxxxv, 1485.

[6] HALL, A. J., and YATES, G., "The Sheffield Outbreak of Epidemic Encephalitis in 1924," *Med. Res. Council Spec. Report*, No. 108, London, 1926, 48.

[7] HOWELL, C. M. H., "Sequelæ of Lethargic Encephalitis," *Brit. Med. Jour.*, 1923, ii, 1086.

[8] JELLIFFE, S. E., "Somatic Pathology and Psychopathology at the Encephalitis Crossroad: a Fragment," *Jour. Nerv. Ment. Dis.*, 1925, lxi, 561.

[9] *Ibid*, "Postencephalitic Respiratory Syndrome: Phenomenology and Pathologic Considerations," *Arch. Neur. and Psychiat.*, 1927, xvii, 627.

[10] *Ibid*, "Postencephalitic Respiratory Disorders," New York, 1927.

[11] LÉVY, G., *Thèse de Paris*, 1922, No. 439, quoted in her monograph: "Les manifestations tardives de l'éncéphalite épidémique," Paris, 1925, 252.

[12] MARIE, P., and LÉVY, G., "Deux manifestations particulières de l'encéphalite épidémique prolongée. Forme respiratoire: forme insomnique," *Rev. neurol.*, 1922, xxxviii, 1233.

[13] RUNGE, "Psychopathie u. chronische Encephalitis Epidemica mit eigenartige Symptomatologie (larvierte Onanie)," *Arch. f. Psychiat.*, 1923, lxviii, 429.

[14] TURNER, A., and CRITCHLEY, M., "Respiratory Disorders in Epidemic Encephalitis," *Brain*, 1925, xlviii, 72.

[15] WIMMER, A., "Chronic Epidemic Encephalitis," London, 1924.

[16] WITZEL, A. E., "Epidemic Encephalitis and the Psychoneuroses," *State Hosp. Quart.*, 1925, x, 387.

[17] HALL, G. W., MAYERS, L. H., and KERR, J. A., "Diabetes Insipidus as a Sequel to Epidemic Encephalitis, A Study of the Water and Chloride Balance in Two Cases," *Jour. of Neurol. and Psychopath.*, 1925, vi, 181.

TABLE I.

THE OUTCOME OF THE RESPIRATORY DISORDERS CORRELATED WITH THE CHARACTER OF THE BREATHING ANOMALY.

(The letters A, B, etc., refer to the groups considered in the text.)

Variety of Disorder.	Case No.	Time (in months) over which observed.				
		A	B	C	D	E
TACHYPNŒA	2	32	—	—	—	—
	3	—	32	—	—	—
	4	—	31	—	—	—
	7	—	24	—	—	—
	8	—	42	—	—	—
	9	—	32	—	—	—
	10	—	27	—	—	—
	11	—	35	—	—	—
	13	—	—	31	—	—
	17	—	—	—	27	—
	18	—	—	—	31	—
	21	—	—	—	36	—
	23	—	—	—	15	—
	25	—	—	—	9	—
	27	—	—	—	16	—
	29	—	—	—	—	37
BRADYPNŒA	5	—	31	—	—	—
BREATH-HOLDING	1	36	—	—	—	—
	14	—	—	18	—	—
	16	—	—	—	12	—
	20	—	—	—	30	—
	28	—	—	—	—	5
YAWNING	15	—	—	—	31	—
	19	—	—	—	16	—
	26	—	—	—	6	—
BLOWING, SNIFFING	6	—	18	—	—	—
	12	—	23	—	—	—
	22	—	—	—	30	—
COUGHING	24	—	—	—	15	—

TABLE II.

The Parkinsonian Syndrome and its Association with Respiratory Disorders.

		Time (in Months) over which observed.				
		A	B	C	D	E
With Parkinsonism.	Mild	—	18	—	—	—
		—	—	—	15	—
		—	42	—	—	—
		—	—	—	5	—
	Moderate	—	32	—	—	—
		—	26	—	—	—
		—	24	—	—	—
		—	32	—	—	—
		—	27	—	—	—
		—	35	—	—	—
		—	—	18	—	—
		—	—	—	36	—
		—	—	—	39	—
		—	—	—	16	—
		—	—	—	31	—
		—	—	—	6	—
	Severe	—	—	—	16	—
		—	—	—	—	37
		—	—	31	—	—
		—	31	—	—	—
Total		—	9	2	8	1
Without Parkinsonism		36	—	—	—	—
		32	—	—	—	—
		—	23	—	12	—
		—	—	—	27	—
		—	—	—	31	—
		—	—	—	30	—
		—	—	—	9	—
		—	—	—	—	5
Total		2	1	—	5	1

TABLE III.

CORRELATION OF RESPIRATORY WITH BEHAVIOUR DISORDERS.

Type of Case.	Time (in Months) over which observed.				
	A	B	C	D	E
WITH CONDUCT OR PSYCHOTIC CHANGES.	36	32	—	15	—
	—	24	—	36	—
	—	42	—	—	—
	—	32	—	—	—
	—	23	—	—	—
TOTAL	1	5	—	2	—
WITHOUT CONDUCT OR PSYCHOTIC CHANGES	32	31	31	31	5
	—	26	18	12	37
	—	18	—	27	—
	—	27	—	31	—
	—	35	—	16	—
	—	—	—	30	—
	—	—	—	30	—
	—	—	—	15	—
	—	—	—	9	—
	—	—	—	16	—
TOTAL	1	5	2	11	2

TABLE IV.

SHOWING THE YEAR OF THE ORIGINAL ATTACK IN THE PRESENT SERIES OF CASES. OFFICIAL STATISTICS ARE ALSO APPENDED, GIVING THE TOTAL NUMBER OF NOTIFICATIONS AND DEATHS OCCURRING IN ENGLAND AND WALES.

Date of Original Illness.	Number of Cases.						Figures for England & Wales.	
	A	B	C	D	E	Total.	Notifications.	Deaths.
1919 ..	—	—	—	—	—	0	541	294
1920 ..	—	—	—	1	—	1	890	480
1921 ..	—	—	—	—	—	0	1,470	729
1922 ..	—	1	—	—	—	1	454	339
1923 ..	—	1	—	—	—	1	1,025	531
1924 ..	2	8	2	9	2	23	5,039	1,407
1925 ..	—	—	—	3	—	3	2,635	1,372
1926 ..	—	—	—	—	—	0	2,267	1,324

A REMARKABLE CASE OF PACHYMENINGITIS HYPERTROPHICA PRESENTING SPINAL BLOCK AND FROIN'S SYNDROME.

By D. WIERSMA, Groningen, Holland.

INTRODUCTION.

In another paper[24] I published the histories of two patients in whom an obstruction of the spinal canal, caused by tumour, could be demonstrated by the existence of Froin's syndrome (xanthochromia and massive coagulation of the cerebrospinal fluid). In both cases the presence of spinal block was confirmed by the result of röntgenography. In the first patient injection of air by means of lumbar puncture led to an exact determination of the level of the tumour, as the air was seen to be confined underneath the obstruction and to form a distinctly less opaque column, which reached as high as the ninth thoracic vertebra. At the operation an extramedullary intradural fibroma, which was seated at this level, was removed and afterwards the patient recovered nearly completely, presenting only a very small remainder of the paraplegia, which for several months before the laminectomy had been a total one. In the second patient lipiodol was used as a contrast object: it was injected by the lumbar route, the patient's pelvis being raised. It stopped at the level of the tenth thoracic vertebra, which when laminectomy was performed appeared to be affected by a sarcoma. Neither the operation nor X-ray treatment, which was then tried, had any success. Without having presented even a temporary improvement of the total transverse lesion of his spinal cord, the patient died a few months after the operation.

When in 1903 Froin for the first time called attention to xanthochromia and massive coagulation of the cerebrospinal fluid, he thought that the syndrome was found only in cases of tumour either of the spinal cord itself or of its membranous or bony surroundings. But later on it appeared to develop sometimes in cases of spinal block of the most varied kind. Mingazzini[11] mentions its presence in Pott's disease, in luxation or fracture of the vertebræ and even in cases where no real blocking of the spinal canal exists. Babonneix and Voisin[1] emphasize its frequency in pachymeningitis hypertrophica cervicalis. This disease consists in a hypertrophic inflammation of the dura mater, which owing to the extreme degree of thickening of that membrane in its progression always leads to a total obstruction of the spinal canal. Though the inflammatory process usually has its localisation at the level of the cervical enlargement, it may be found in any other part of the spinal cord and in that case gives rise to very uncommon clinical symptoms, so that the diagnosis becomes very difficult or even impossible. The spinal cord itself rarely remains unaffected:

From the University Clinic of Psychiatry and Neurology in Groningen, Director Prof. E. D. Wiersma.

in many cases holes in its substance are seen, which much resemble the anatomical changes found in syringomyelia. The pathogenesis, however, of these cavities is not known as yet, though Müller[12] mentions that it is probably not the same as in ordinary syringomyelia.

The following case, which is interesting from its many clinical and anatomical peculiarities and by the fact that it presented Froin's syndrome, may be put on record.

CASE HISTORY.

M.d.V., postman, age 22 years, came into the clinic in the beginning of March 1924. In his previous history nothing remarkable was found.

Present Illness.—About the middle of January, 1924, he was aware of a slight numbness of the toes, first in his left foot and a few days after in his right. Soon the numbness, connected with a prickling sensation, rose to the ankles, the knees and even to the gluteal region, but he could do his work well and did not pay much attention to his troubles. In the beginning of February he suddenly got a sharp pain in his back and his chest, which was a little better when he was walking. At the end of the month he began to complain of severe sphincter troubles: he could no longer pass urine spontaneously and had to be catheterised. Defæcation too was difficult. A few days later the patient could not get up from his bed: the legs had become progressively more and more powerless, though the pains in back and chest became rather less.

Somatic examination did not reveal any deviation in any of the internal organs. The urine contained some leucocytes but no albumen.

Examination of the vertebral column and nervous system.—Mobility of the vertebral column rather good; no deformation. Pressure on the fifth dorsal spinous process, and to a less degree on the sixth, was painful. Skull and cranial nerves did not present anything abnormal, nor did the arms. The muscle strength of the trunk was reduced; the upper abdominal reflexes were diminished, the middle and inferior reflexes absent. Both legs were paretic; the muscle tonus was very poor; the kneejerks and Achillis tendon reflexes were diminished; Babinski's reflex was not obtained. Sensation was diminished for all qualities below the groins, so that for tactile and painful stimuli a total anæsthesia, for thermal stimuli a hypæsthesia was found. Above this field of diminished sensation, up to the umbilicus, a marked hyperæsthesia and hyperalgesia were present, but at a higher level an anæsthetic zone of about 10 cm. breadth was present again, which was limited both above and below by a small zone of hypæsthesia. Above this zone a field of hyperæsthesia and hyperalgesia existed, extending as far as the third rib.

During the next few days the patient had complete retention. The urine was removed twice a day by catheter. He was somewhat feverish, but did not present alarming symptoms. In X-ray photographs of the vertebral column nothing abnormal was seen. After a week both legs had become totally paralysed; the hyperalgetic field on the abdomen disappeared. A few days after the patellar and Achillis tendon reflexes were no longer elicited. The upper abdominal reflexes too had disappeared.

The diagnosis of a transverse lesion of the spinal cord was readily made, but it was more difficult to determine its nature. The rather acute though insidious beginning, the rapid progression, the ascending character of the sensory level, and the fever seemed to point to a myelitis. A vertebral lesion could not be diagnosed, though the painfulness of the fifth dorsal spinous process perhaps seemed to point in that direction, for the X-ray photographs of the vertebral column did not reveal anything that could be regarded as a sign of disease. Besides, the flaccid paraplegia of the legs could not well be explained

if there was only a disorder in the cervical and upper dorsal cord : it must have been seated at a much lower level as well. A tumour therefore did not seem very probable, but it was felt that lumbar puncture might point more in that direction.

The latter was performed because the patient began to complain of pain in his neck and presented some neck rigidity. The pressure of the cerebrospinal fluid was low ; the fluid itself was distinctly yellow and clear, and within two hours it presented massive coagulation. Pandy's and Nonne's reactions were highly positive ; no sugar appeared to be present. The fluid contained very few erythrocytes and a single leucocyte. The yellow colour appeared not to originate from hæmoglobin, but from lutein. No micro-organisms could be cultivated on ascites-agar. A second lumbar puncture a week after gave the same result.

Gradually formication in both hands developed and the muscle strength of the arms diminished somewhat. Now and then attacks of hiccough occurred, which were very distressing. They were, however, rather easily controlled by the administration of $\frac{1}{4}$ mgr. of atropine sulphate.

It was now clear that an obstruction of the spinal canal was highly probable. As no vertebral lesion of any kind was present, it was considered to be due either to a tumour or to an inflammatory process within the spinal canal itself. In the last few weeks the condition had progressed and the motor as well as the sensory troubles had reached the arms. This course seemed to speak in favour of an inflammation, for it is not easy to see how a tumour can give rise to such a development on the clinical side. But a mere myelitis on the other hand is not likely to cause an obstruction of the spinal canal. So the uncertainty was not done away with and the further clinical course did not bring much more light.

The patient gradually got worse and the attacks of hiccough became more and more frequent. By the end of March the hyperalgesic zone had reached the clavicle and only a few cm. lower a total anæsthesia and analgesia existed. From the beginning of April the patient had nearly every day a rigor with a rise of temperature to 40°, 41° or even 42° C. Between those acute elevations the temperature fell again to the normal level. The hiccough in the meanwhile became more and more intractable towards the end of April drugs no longer influenced it. In the beginning of May, 1924, he died.

The autopsy was made by M. Mieremet. In the region of the lesser curvature of the stomach a large ulcer was found. It appeared to be very recent, so that it could not have any bearing on the etiology of the spinal cord affection. Apart from a purulent cystitis and pyelonephritis, which were to be expected, no disturbances of the internal organs were present.

The spinal cord was very difficult to remove ; in the dorsal region it was very soft and adherent to the dura, and could not be separated without tearing the pia. A yellowish mass protruded which gave the impression of being pus. Microscopical investigation, however, showed that it did not contain leucocytes or microorganisms, but was only an indefinable mass of detritus. When incisions were made at various levels of the spinal cord a cavity appeared to be present in the deepest part of the left dorsal column which as far as could be made out microscopically did not communicate with the central canal. It extended from the third cervical segment to the lumbar region, though in the dorsal cord it could not be traced because of the total destruction there present. The white matter in the dorsal and lateral columns in the cervical and lumbar segments seemed to be somewhat degenerated. The meninges were much thickened.

M. Mieremet kindly gave some pieces of the spinal cord to the laboratory of our clinic, where they were cut and stained with hæmatoxylin-eosine and by Van Gieson and Weigert-Pal methods for microscopical investigation. The meninges appeared to be highly thickened and to consist for the greater part of granulation tissue in which many exudation cells and a marked hyperæmia pointed to the inflammatory character of the process. No gummata or endarteritis could be found.

DISCUSSION.

Thus a diagnosis of pachymeningitis hypertrophica had to be made. This affection is extremely rare and very few elaborate descriptions of it are known. Müller[12] says: "Leider sind bisher nur ganz vereinzelte Fälle ätiologisch, klinisch und histologisch mit moderner Methodik untersucht, vor allem mit Hilfe der Lumbalpunktion und der Komplementbindungsreaktion auf Syphilis." As in this case all these methods of investigation have been applied, I believe it may contribute a little to increase of our knowledge of the disease.

I.—ETIOLOGY.

As far as etiology is concerned, it may be taken as an example and proof of the fact that syphilitic infection is by no means essential in the production of the disease. For no signs of this condition were found, either in the history or in the actual clinical state of the patient: Wassermann's reaction in the blood serum was negative, while in a xanthochromic and highly albuminous cerebrospinal fluid it is of no value. In both cases of tumour mentioned above it gave doubtful results. One cannot, therefore, regard as an omission the fact that it was not performed in the fluid in this case. Microscopically no syphilitic alterations could be demonstrated.

Gross[8] mentions a case of pachymeningitis hypertrophica of tuberculous nature, but he emphasizes the rarity of a localisation only in the meninges without the slightest affection of the vertebræ. Still, tuberculosis is one of the most well-defined etiological factors of the disease known. Rümke and Goudsmit[15] report a case in which the inflammation was also probably due to a tuberculous infection, though no tubercles could be demonstrated in a piece of dura removed at operation. Other factors mentioned by various authors are, according to Cassirer[6], cold, overstrain, trauma, alcohol: and, according to Babonneix and Voisin[1] alcohol, lead poisoning, rheumatism: but in most cases syphilis seems to be the main cause. It was so, *e.g.*, in a case of Souques, Blamoutier and de Massary[19], which presented a positive Wassermann's reaction in blood serum and cerebrospinal fluid while the patient recovered almost completely after treatment with bismuth preparations. A very remarkable case is reported by Weigeldt[22]. It concerns a patient who, more than two years after production of lumbar anæsthesia (novocaine) for a pelvis operation, developed a total paraplegia of the legs. He died and the autopsy showed a complete obliteration of the lumbar theca, which had in all respects the appearance of a pachymeningitis hypertrophica. Weigeldt knows of three other patients in whom a lumbar anæsthesia was followed by obliteration of the subdural space and of two in whom an intradural injection of neosalvarsan had the same result. All these patients recovered: the obliteration was determined by means of lumbar puncture. The last two cases are less instructive, for here the syphilis of course may have been the cause of the affection.

From all the various etiological factors which have been demonstrated or supposed we may conclude that irritation, by preference chronic, of the spinal meninges mainly contributes to the beginning of the disease. In our case we have to ascertain if a chronic irritation of any kind can be demonstrated. Other evidence of it than a very busy life, however, is not obtainable. Possibly the great amount of walking that the patient had done contributed somewhat to irritation of the spinal meninges.

II.—SYMPTOMATOLOGY.

The clinical aspect of this case was different from the usual. In most cases the alterations are localised entirely or mainly in the cervical enlargement and this localisation gives rise to rather typical symptoms, which were first described by Charcot and Joffroy. The first stage is one of bilateral root pains and paræsthesiæ in the arms and is followed by a stage of segmental paralysis in which flaccid paralysis of the arms occurs with typical attitudes of the hands (*mains de prédicateur*) because of the affection mainly of the roots of origin of ulnar and median nerves, and finally by a stage of affection of motor tracts, producing a spastic paralysis of the legs. The authors gave the name of pachymeningitis hypertrophica cervicalis to the disease, as no other localisation was known to them. Subsequently it appeared, however, that other localisations did occur. Cassirer[6], Babonneix and Voisin[1] and Müller[12] mention the possibility of localisation in any part of the spinal cord and they emphasize the difficulty of making the diagnosis when the affection is seated in the dorsal segments. Confusion with tumour is nearly inevitable, especially when no distinct bilateral root pains are present, as in our patient. Westerhuis[23] mentions a case in which by means of 'lipiodol ascendant' and 'lipiodol descendant' he demonstrated an obstruction of the spinal canal the inferior pole of which was seated at the level of the tenth and the upper at the level of the sixth thoracic vertebra, without being able to determine the nature of the process. This was only possible after microscopical investigation of a small piece of tissue removed at operation. The laminectomy did not bring about any improvement.

In our case the existence of an obliterating process involving several segments was also clear enough, and for this reason an operation was thought useless. But before the autopsy the correct diagnosis was not made. We apparently had to deal with an ascending affection: the movement upward of the sensory level and the appearance in the course of the disease of slight pareses of the arms proved this. The hiccough attacks must probably be regarded as a symptom of irritation, the pathological process finally having reached the fourth cervical segment, which innervates the diaphragm. As the fever was of a septic type it seemed to point to an infective disease. It will perhaps be possible to make the diagnosis of a pachymeningitis hypertrophica in the future, when an obliterating process is found that extends over many segments and presents signs of ascending or descending and a temperature course that points to an infection.

Finally, I wish to discuss a peculiarity of the paraplegia. A glance at Fig. 5 will make clear that a total interruption of the spinal cord in the dorsal part was present. The existence therefore of a flaccid paraplegia of the legs without an affection of the anterior horns in the lumbar segments is not surprising. It is on the contrary so common, that it has given rise to the conception of Bastian's so-called law, according to which a flaccid paraplegia necessarily should follow a total interruption of the spinal cord. Brouwer[1], on the contrary, having shown that during the long continuance of such an interruption the tendon reflexes may come back and spasticity appear, thinks that in those cases which at the beginning seem to follow Bastian's law we are dealing with a process of diaschisis, which after a shorter or longer time will disappear. Several other authors have recorded cases in which a total interruption in the dorsal or cervical segments of the spinal cord was followed by only temporary loss of reflexes. I am inclined to believe, therefore, that in our patient the affection had not lasted long enough for the diaschisis phenomena to disappear, but that, had he lived longer, the tendon reflexes of the legs would have returned.

III.—HISTOLOGY.

Only the histological aspect of the case is left for discussion and for that purpose I shall give a more detailed description of the microscopical preparations than I did when mentioning the results of the autopsy.

In the upper part of the cervical cord the first thing that strikes the eye is the cavity already alluded to. It really appears neither to communicate with the central canal nor to have a wall of its own, but to be surrounded by a zone of necrotic tissue that occupies a part not only of the posterior columns but of the posterior horns as well (Fig. 1). This

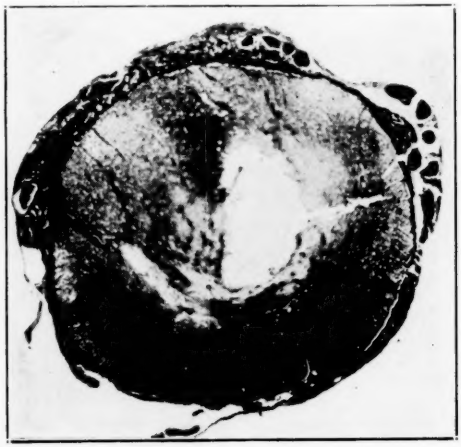


Fig. 1. Upper part of cervical cord. Stain: Van Gieson.

tissue consists of hardly recognizable debris with indistinct cell nuclei and here and there a single lymphocyte. Further examination of the section shows that the ganglion cells of the anterior horns are rather well conserved. Here and there in the gray as well as in the white substance some small infiltrations consisting of mononuclear cells are to be seen. The white substance presents a marked degeneration in all the columns, least noticeable in the anterior pyramidal tracts. As to the meninges, they appear to be much thicker than in normal circumstances and in places fill up the whole subdural space. They consist of a vascular and hyperæmic granulation tissue in which numerous fibroblasts, plasma cells and lymphocytes are to be seen. At some points obliterated blood vessels filled with fibroblasts can be distinguished and at others there are small hæmorrhages. Layers of older connective tissue can in some places be made out (Fig. 2).

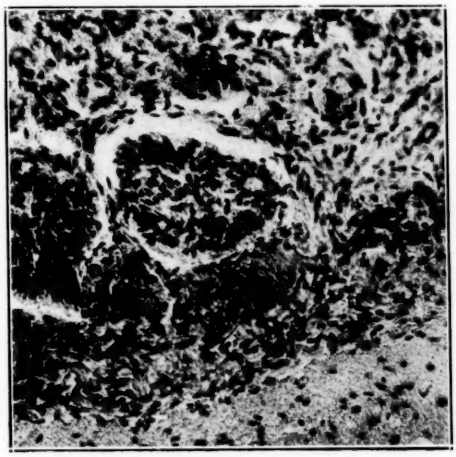


Fig. 2. Part of the granulation mass in the meninges of about the same section as fig. 1. Stain: hæmatoxylin-eosine.

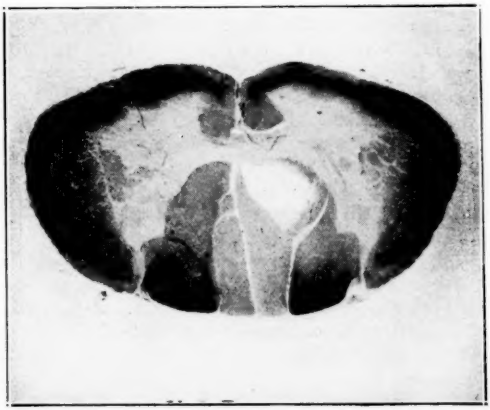


Fig. 3. Inferior part of cervical cord. Stain: Weigert-Pal.

Lower in the cervical cord the necrosis round the cavity is less extensive, though as distinct as in the former sections. The cell nuclei have disappeared almost entirely, an occasional small round nucleus being seen amid the tissue debris. Especially in the neighbourhood of the necrotic region small infiltrations are visible, consisting of mononuclear cells (Fig. 4). Here, too, the white substance presents a considerable degeneration

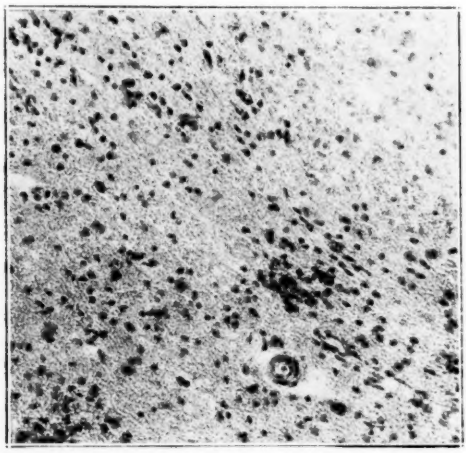


Fig 4. Infiltration of mononuclear cells in the neighbourhood of the necrotic tissue in the same part as fig. 3. In the right upper corner of the figure a part of the necrotic tissue surrounding the cavity. Stain : haematoxylin-eosine.

in all the columns (see Fig. 3). The meninges are less thickened at this level, but apart from that have quite the same aspect as in higher sections. The same granulation tissue with fibroblasts, plasma cells and lymphocytes is found here, and also a strip of older connective tissue at one or two places.

The middle segments of the thoracic region present the greatest destruction possible (Fig. 5). In the sections of this region nothing can be distinguished of the proper spinal cord substance: it is reduced to a necrotic and indefinable mass of tissue fragments, much of which has fallen out during the preparation of the section. The meninges are greatly thickened, their internal layer is necrotic and hardly distinguishable from the spinal cord substance, but gradually the granulation tissue becomes recognizable in the outer layers and it appears to be of the same nature as in sections at a higher level. Hyperaemia, fibroblasts and lymphocytes are present here as well, and strips of older connective tissue are not missing. The external layer of the section consists of membranous connective tissue tightly matted together with the granulation mass in the subdural space.

The lumbar part of the spinal cord appears to be destroyed to a much less extent (Fig. 6). It looks much like what was found in the cervical segments. The same cavity in the posterior columns can be distinguished, filled with necrotic debris and without the well defined limits to the zone of necrosis that in the cervical cord were seen to surround it. Here and there a small infiltration of mononuclear cells is found (Fig. 7). The ganglion cells of the anterior horns are perfectly well conserved. Degeneration is present in

Fig. 5. Thoracic section of the spinal cord. Stain: Van Gieson.

Fig. 6. Lumbar section of the spinal cord. Stain: Van Gieson.

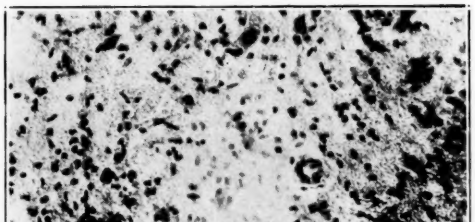


Fig. 7. Mononuclear infiltrations in the lumbar segments. Stain: Van Gieson.

all the columns. The meninges have the same aspect as in the other sections. Though not as much thickened, they consist of quite the same granulation tissue, interrupted in some places by a strip of older connective tissue.

IV. —CAVITY-FORMATION.

The description of the microscopical preparations may have made clear that though a general resemblance to syringomyelia may be found in the existence of a lengthy cavity almost throughout the whole extent of the cord, it can be distinguished from genuine syringomyelia very well. For in our case the cavitation does not originate from a primary gliosis arising in the region of the central canal, as is usually the case in ordinary syringomyelia, but takes its origin in decay of tissue, as may be concluded from the zone of necrosis that is found to surround it everywhere and from the fact that it is filled with necrotic fragments in the lumbar segments. The occurrence of cavities in cases of pachymeningitis hypertrophica is mentioned by several authors and they all ascribe it to necrosis. But the cause of the necrosis is not easy to determine. It is not probable that propagation of the inflammatory process from the meninges to the spinal cord substance should be made responsible for it, for, in the first place, we cannot understand why the inflammatory spread should be confined in the greater part of the cord to a small region almost in the middle of the section: we should expect it on the contrary in a marginal zone. And in the second place the cavity and its surroundings present very few signs of inflammation: except for some small infiltrations here and there, only uncomplicated necrosis can be found. It seems much more probable that the necrosis must be ascribed to want of blood supply and that it is of ischæmic nature. In the meningeal granuloma many obliterated blood vessels are present, so it is not surprising that the blood supply of the cord has become

deficient. According to Pitres and Testut[14] the cavity lies exactly in the region of the terminal ramification of the posterior peripheral artery, which is a branch of the perimedullary arterial circle found in each segment. I admit it is not immediately comprehensible why in every segment the same region must have become ischæmic, since each has its own arterial supply. Perhaps the consideration that the region of the terminal ramification of the posterior peripheral artery is one of the parts farthest from the supplying arterial circle may contribute to the explanation of the facts. For when by the blocking of afferent and perhaps also of efferent blood vessels the blood-pressure in the supplying arterial circle is lowered, those parts will necessarily first be deprived of blood in which the arterial pressure has been lowest from the beginning. Now the lowest arterial pressure is found in the arteries which are most remote from the supplying branch.

Concerning the point of origin of the inflammation, recent authors differ from Charcot and Joffroy. The latter thought that the affection originated in the dura mater, but Müller[12], Cassirer[6] and Babonneix and Voisin[1] suggest that in many cases it arises in the leptomeninges or even in the superficial layers of the cord itself and spreads only secondarily to the dura mater. From study of our preparations we may reach the same conclusion, for in all the sections the greatest anatomical changes were found between the dura and the cord, whereas the dura itself, as well as the cord substance, shows distinct signs of inflammation only in the thoracic region. Most of the changes in the spinal cord are due to necrosis as a consequence of deficient blood supply.

V.—SYNDROME OF FROIN.

Since this case, as well as the cases of tumour mentioned at the beginning of the paper, showed xanthochromia and massive coagulation of the cerebrospinal fluid, it may be of interest to conclude with a short discussion of the significance of this syndrome. Froin thought it was a reliable sign for the diagnosis of blockage, but it has also occurred in patients in whom no process obliterating spinal passages has been found. De Sanctis[16] mentions the occurrence of the syndrome, for instance, in various kinds of meningitis. I saw it myself in a young man who had abruptly become ill, presenting the aspect of an acute catatonia, but after three days neck-rigidity and Kernig's sign developed. A lumbar puncture was made: the cerebrospinal fluid was intensely yellow and coagulated within 15 minutes. It did not contain red blood corpuscles and only 3 white blood corpuscles per cubic mm. The globulin reactions were strongly positive. The next day he died; at the autopsy the existence of tuberculous meningitis was demonstrated.

Though not confined to cases of spinal block, the syndrome of Froin is very rare in other conditions. Its diagnostic value is therefore considerable, if in cases of block it occurs with great frequency. But it is exactly on this point that the opinions of various authors differ. Dandy[7] saw xanthochromia

only once in a series of 36 cases of tumour, Gross[7] three times in 21 patients with spinal block, one of whom only presented massive coagulation of the cerebrospinal fluid at the same time. In agreement with most German authors he thinks the syndrome very rare and therefore of much less value than Nonne's syndrome—a large amount of globulin coupled with a normal or subnormal number of cells. He found it 13 times in his series. Bingel[2, 3] however, in three cases of tumour found xanthochromia and massive coagulation in one and in two a perfectly normal fluid. In a case of caries with spinal block he found xanthochromia but no coagulation, while the globulin reaction was feeble. French authors on the contrary find the xanthochromia syndrome much more frequently. Guillain, Alajouanine, Mathieu and Bertrand[9] in a case of cauda perithelioma; Guillain, Alajouanine, Périsson and Petit-Dutaillis[10] in a case of fibroglioma of the eleventh dorsal segment mention its occurrence, and curiously enough they found in both cases xanthochromic cerebrospinal fluid above the tumour, though in the last case it was present below the growth as well. Souques, Blamoutier and De Massary[19] found the syndrome in pachymeningitis hypertrophica. Babonneix and Voisin[1] say in this last disease it is nearly always present. In Dutch literature xanthochromia is mentioned in three cases of tumour, described by Brouwer and Oljenick[5], by Pameyer[13] and by Von Ziegenweidt[25], but in Pameyer's case it was not combined with massive coagulation. In six cases described by Dutch authors (another case of Brouwer and Oljenick[5], De Vries[21], Stenvers[20], Pameyer's second case[13] and two cases of Sillevis Smitt and Bok[17, 18]) the syndrome was entirely absent. Nonne's syndrome was present in four of these cases and in Westerhuis' case of pachymeningitis hypertrophica[23]. But in two cases of tumour the cerebrospinal fluid was almost normal. Together with personal cases I have collected the following data relating to cerebrospinal fluid syndromes in spinal block patients.

Nonne's syndrome	18 cases.
Xanthochromia	15 cases.
Normal or almost normal fluid	4 cases.

I consider that xanthochromia is frequent enough to have considerable diagnostic value, although Nonne's syndrome seems to be a little more common.

I wish here to point out that according to De Sanctis[16] the value of xanthochromia remains the same whether massive coagulation is present or not, for when absent the latter usually appears when one drop of blood serum is added. The coagulation is due to a certain amount of fibrin and it cannot appear when no fibrin ferment is present. By means of a drop of serum this ferment is added and at once the fluid coagulates.

The yellow colour must not be ascribed to hæmoglobin, since this substance is often missing in xanthochromic fluid, but to lutein, the same colouring matter that causes the yellowish tinge of blood serum; it can always be demonstrated in the cerebrospinal fluid when xanthochromia is present. It has been found in all our patients. As to the cause of the syndrome Babonneix and Voisin[1]

think it must be sought in the lowered pressure of the fluid below the block. This causes a local oedema and transudation of blood plasma into the dural theca. I believe, however, this theory is incorrect, as the xanthochromia may be found above the obstruction and in cases where no spinal block is present at all. It is more reasonable to make irritation of the meninges responsible for it, as a result of which they become more permeable for components (fibrin and lutein) of the blood plasma.

REFERENCES.

[1] Babonneix and Voisin, "Méningites chroniques," *Traité de pathologie médicale*, 1925, v, 606.

[2] Bingel, "Intralumbale Lufteinblasung zur Höhendiagnose intraduraler und extraduraler Prozesse und zur Differentialdiagnose gegenüber intramedullären Prozessen," *Deut. Zeits. f. Nervenheilk.*, 1921, lxxii, 359.

[3] Bingel, "Diagnostische Vorzüge der Lumbalpunktion unter Gaseinblasung (ohne Berücksichtigung des Röntgenverfahrens)," *Deut. Zeits. f. Nervenheilk.*, 1921, lxxv, 155.

[4] Brouwer, "Über Querläsion des Rückenmarks beim Menschen und das Bastiansche Gesetz," *Psychiat. en neurol. Blad.*, 1915, xix, 376.

[5] Brouwer and Oljenick, "Lipiodol-test in tumours of the spinal cord," *Acta psychiat. et neurol.*, 1926, i, 15.

[6] Cassirer, "Pachymeningitis cervicalis hypertrophica," Oppenheim's *Lehrbuch der Nervenkrankheiten*, 1923, i, 420.

[7] Dandy, "The diagnosis and localisation of spinal cord tumours," *Ann. of surgery*, 1925, lxxxi, 223.

[8] Gross, *Klinische und Liquordiagnostik der Rückenmarkstumoren*, 1925.

[9] Guillain, Alajouanine, Mathieu and Bertrand, "Sarcome périthélial de la queue de cheval," *Revue neurol.*, 1924, i, 513.

[10] Guillain, Alajouanine, Périsson and Petit-Dutaillis, "Diagnostic d'une tumeur intra-rachidienne," *Revue neurol.*, 1925, i, 14.

[11] Mingazzini, "Ein neuer Beitrag zur örtlichen Diagnostik der Rückenmarkstumoren," *Deut. Zeits. f. Nervenheilk.*, 1925, lxxxiv, 45.

[12] Müller, "Pachymeningitis hyperplastica interna," v. Bergmann and Staehelin's *Handbuch d. inneren Medizin*, 1925, v. 800.

[13] Pameyer, "Twee gevallen van intravertebraal extraduraal gezwel," *Ned. Tijdschr. v. Geneesk.*, 1917, lxi(i), 816.

[14] Pitres and Testut, "Artères de la moelle épinière," *Les nerfs en schémas*, 1925, 533.

[15] Rümke and Goudsmit, "Over heelkundige behandeling der pachymeningitis cervicalis hypertrophica," *Ned. Tijdschr. v. Geneesk.*, 1927, lxxi(i), 2854.

[16] De Sanctis, "Klinischer Beitrag zum Studium der Rückenmarksgeschwülste und des Froinschen Syndroms im Liquor," *Deut. Zeits. f. Nervenheilk.*, 1923, lxxvi, 183.

[17] Sillevis Smitt and Bok, "Lipidol bij het Röntgenologisch onderzoek van het ruggemerg en zijn vliezen," *Ned. Tijdschr. v. Geneesk.*, 1924, lxviii(i), 2213.

[18] Sillevis Smitt and Bok, "Lipidol bei der röntgenologischen Untersuchung des Rückenmarkes und seiner Häute," *Deut. Zeits f. Nervenheilk.*, 1925, lxxxvii, 263.

[19] Souques, Blamoutier and De Massary, "Injection lipiodolée sousarachnoidienne dans un cas de pachyméningite cervicodorsale. Arrêt total du lipiodol dans la région cervicale inférieure," *Revue neurol.*, 1924, i, 6.

[20] Stenvers, "Twee intradurale, extramedullaire gezwellen," *Ned. Tijdschr. v. Geneesk.*, 1917, lxi(ii), 863.

[21] Ernst de Vries, "Een geval van ruggemergsgezwel," *Ned. Tijdschr. v. Geneesk.*, 1915, lix(i), 1525.

[22] WEIGELDT, "Rückenmarksschädigungen nach Lumbalanaesthesien," *Deut. Zeits. f. Nervenheilk.*, 1925, lxxxiv, 121.

[23] WESTERHUIS, "Pachymeningitis hypertrophica dorsalis," *Ned. Tijdschr. v. Geneesk.*, 1927, lxxi(i), 3194.

[24] D. WIERSMA, "Clinical and anatomical experiences in tumour of the spinal cord," *Acta psychiat. et neurol.*, 1927, ii.

[25] VON ZIEGENWEIDT, "Een geval van gezwel van het ruggemerg," *Ned. Tijdschr. v. Geneesk.* 1915, lix(i), 238.

EPILEPTIC VARIANTS.

By S. A. KINNIER WILSON, London.*

In the course of twenty years' hospital and private practice there have come under notice a large number of cases which present intimate or remote similarities to the phenomena of epilepsy as usually understood, but which nevertheless do not seem to resemble each other, at least not superficially. Unfortunately, in many instances one has to rely on the description furnished by either the patient or his friends (there is no reason, of course, to doubt these descriptions are veridical), since development of the symptoms concerned during the period of consultation and examination can scarcely be expected, helpful though it would prove. Close study of these conditions, which may conveniently be termed epileptic variants, is particularly fruitful in dispelling from the mind any idea of epilepsy as a self-contained and sharply delimited clinical entity. I pick up a recent book on neurology for practitioners and therein I find the chapter on epilepsy opens with the dictum: "Epilepsy is a dreadful disease." Five words can hardly contain more than a similar number of errors; these five unquestionably embody two, for epilepsy cannot be regarded as a disease, and experience teaches that the majority of cases are certainly benign. We have laboured for years under the unenlightening procedure of elevating symptoms into diseases, giving them names, and forcing new clinical instances into old nosological conceptions. This has been notoriously the case with epilepsy; so stereotyped is the idea in the minds of many that it is a sinister "disease" that to any given case of "fits" this conception is forthwith transferred—in the absence, that is to say, of accompanying symptoms which may point otherwise. Against this mental habit a protest must be raised; one of the best ways of counteracting its harmfulness is by the consideration of epileptic variants.

At the outset it is, perhaps, impracticable to dispense with some kind of exposition of what should be conveyed by the term "epilepsy." In a recent Harveian Lecture,[1] as well as in a paper[2] read before the Assurance Medical Society I have explained at some length the views commending themselves to me in respect of epilepsy and its connotation. Without here repeating general conclusions therein adopted, I should like to restate in a somewhat different form what seems to me the appropriate way of facing the problem.

The clinical phenomena with which we are concerned must be considered physiologically; they represent escape from physiological inhibitions. Whether they indicate only "release," or also "irritation," is immaterial or at least

* British Medica Association Lecture, delivered at a meeting of the Swansea Division, December 8th, 1927.

of secondary importance; if an irritative process is to exteriorise itself it must succeed in overcoming inhibition. Escape of function is no preserve of motor mechanisms, as when the limbs of the patient with spastic paraplegia exhibit involuntary flexor spasms, or when the limbs of the epileptic are convulsed; or as, indeed, may conceivably be the case with movements thought to be "voluntary," since even for the manifestation of these release from transcortical inhibition may be an essential preliminary. In many epileptic phenomena symptoms of another order are apparent—the aura, if present, is a sensation, therefore a psychical thing, a change in consciousness; sensory systems can undergo release, as in sensory Jacksonian epilepsy (in some varieties sensory manifestations may constitute almost the whole fit). Once the process is initiated, it may, or may not, spread; mechanisms, motor or sensory, or both, on the same level, or on lower or higher, may in turn submit and their resistance be overcome, until in a brief period all are affected, and generalisation is then said to take place. Alternatively, the process may be strictly limited, both on motor and on sensory side; the aura, occurring by itself, may constitute the sole symptom and form a larval fit. Again, loss of consciousness, as in numerous instances of *le petit mal*, can develop almost monosymptomatically. Evidently, then, epileptic possibilities are legion; no rigid semiological framework can be fashioned to embrace them all. This becomes still more apparent if we include, as I think we legitimately may, analogous phenomena consequent on excitation or inhibitory release of visceromotor and viscerosensory centres, *e.g.*, those of the medulla. Their clinical types may seem far removed from epilepsy as ordinarily understood, but they can be considered none the less as belonging to the epileptic category.

Frontal, rolandic, parietal, occipital, temporal epileptic syndromes might be distinguished; also mesencephalic, pontine, bulbar, cerebellar, and, it may be, spinal varieties. And cannot similar physiological processes conceivably affect visceral neural plexuses and ganglionic collections? Is "epilepsy" say of the solar plexus an absurdity? I am far from thinking so.

Common sense compels us, if we appropriate this wide conception of the condition, to speak of "the epilepsies," and to disabuse our minds of the idea that one and all must always display a somewhat forbidding aspect. Doubtless in many instances we discover at a glance that the epilepsy is symptomatic, as when fits develop during an attack of cerebral hæmorrhage, in pronounced toxicosis, under conditions of intracranial pressure or with perceptible vascular disease; this fact alone should have taught us to see in all epileptic manifestations the action of mechanisms irritated or released into independent function, and in so-called idiopathic (that is, unknown) epilepsy our search should still be directed towards discovery of the underlying cause, which must exist.

At this point it is perhaps natural to interpose with the objection that the epilepsies would thus be made synonymous with or equivalent to other orders of phenomena with which they do not appear to have much in common. It

is not expedient clinically that they should be classed with tics, for example, or with reflex movements and spinal automatisms, or with hallucinations, and so on ; nevertheless, the aura of an epileptic fit is a hallucination, one or other of its movement-complexes can resemble flexor or extensor spasmodic automatisms, and minor, repetitive varieties are often separable from tic only on prolonged observation. An epileptic head-nodding or drooping movement in children is extremely like a salaam tic. What are to be the criteria whereby differentiation is effected ?

In its crudeness, violence, complexity, disorderliness, the major fit can scarcely be confused with any other syndrome, yet parts of it are neither disorderly, violent, nor crude ; the aura shows none of these qualities, and not a few of the movements are quite " ordinary," *i.e.*, coordinated, and in no sense convulsive. As for Jacksonian epilepsy, its motor display is largely *sui generis* in respect of both the character of the movements and their march, but slight, localised twitching cannot readily be distinguished from some kinds of myoclonus or of tic. Petit mal is recognised with ease because of its intrinsic features in perhaps a majority of instances, yet every practitioner knows what uncertainty surrounds the exact diagnosis in others. To establish standards of universal applicability even in regard to the main classes of epilepsy thus becomes almost a Sisyphean task. All of us may think we know a fit when we see it, but we should have great difficulty in endeavouring to specify the criteria which we as clinicians are using. Features such as recurrence, transient nature, interference with the stream of consciousness, are in no way pathognomonic, though they are probably foremost in the mind whenever we think of " attacks " or " fits " of any sort. The phenomena of the epilepsies edge off by easy gradations from the motor convulsive seizure to psychical, sensory, or visceral symptoms of a highly disparate kind, and at the same time refuse to be separated for any physiological reason. Clinical conceptions ought to be based on physiological unities, and if the same process can rationally be supposed to be in action behind these diverse clinical syndromes they belong to the group. There is no class of symptom in neurology that has suffered more from nosological artificialities than that still called epileptic.

I shall take first the clinical types that are characterised by variations in the motor phenomena, and submit the following classification as possessing clinical usefulness.

I. MOTOR VARIANTS.

1. Myoclonic or regional epilepsy.
2. Epilepsia partialis continua.
3. Tonic epilepsy.
4. Coordinated epilepsy.
5. Inhibitory or akinetic epilepsy.

Myoclonic or Regional Epilepsy.—This particular variant is far more common than is usually imagined. It takes the shape of irregular twitches of a limb in multimuscular groups, and in colloquial parlance is often termed "the jumps." "I had the jumps this morning" is a habitual remark. The occurrence of such jerking movements without loss of consciousness is as distinctive as it is little known. Not much complained of spontaneously, careful inquiry elicits a history of their existence often for a longer or shorter time before the first paroxysmal fit. Indeed, in some instances they form the sole epileptic indication, as in the following case.

Case I. V. S., female, age 18. Since the age of 14 has suffered practically every morning from more or less violent sudden jerks of the arms, sufficient to throw things out of her hands. Affecting both arms indifferently but not synchronously, these jerks make her knock things off the breakfast table or toss a comb or hairbrush across the room. The legs are unaffected. She has never had a fit in her life, whereas one brother and two sisters suffered from convulsions in infancy, and another brother has had fits since the age of three.

Examination reveals an intact nervous system.

Either unilateral or bilateral, they affect some definite region of the body; sudden adduction or extension of one or both arms, and sudden flexion or extension of the trunk, are the commonest movements. A statistical study by Muskens[3] has established their presence at one or other period in no fewer than 103 out of 150 female, and 82 out of 150 male epileptics. A textbook commonplace is to the effect that the familial myoclonus-epilepsy of Unverricht is a great rarity, and the statement is repeated in recent communications (cf. Crouzon and Bouttier[4]), yet my experience does not bear out the contention.

The following example is characteristic:

Case 2. P. P., female, age 15. Has suffered from major fits at long intervals, having had only four in two years. In addition, she shows typical myoclonic jerks of arms and legs in the mornings, throwing things out of her hands or falling suddenly to the floor without a trace of unconsciousness. Examination is negative. A sister also is epileptic, and has myoclonus.

In view of the curious indifference of the average patient to this abnormal condition until or unless major fits develop its early diagnostic significance ought to be impressed on the practitioner of medicine.

Epilepsia partialis continua.—A second motor variant, doubtless somewhat rare, was described originally by Koshewnikow[5], as long ago as 1894, and has received but scant attention notwithstanding its physiological interest. It differs from the general myoclonic type in that the twitching is limited to one segment of the body, nearly always a peripheral part such as the wrist and fingers, is practically continuous between the paroxysmal fits, and on the whole partakes of the form less of movements than of irregular, individual, muscular contractions. Among those who have reported cases are Orlowski[6], Chorosko[7] (who suggested the term "polyclonia epileptoides continua"), Spiller[8], and Bruns.[9] I have seen several typical cases, of which one is here briefly related.

CASE 3. E. W., male, age 30. Has suffered from epilepsy since childhood, having on an average several fits every week. For about 15 years he has complained of irregular twitches of the right hand and fingers, more or less continuous. They are jerky and abrupt, producing usually flexion but sometimes also extension of the fingers, and either flexion or extension of the wrist. Quite distinct from tremor, they are equally separable from both chorea and athetosis mainly because of their twitching character; in this respect they are classifiable with the myoclonias. The major fits begin in this right hand, becoming generalised with great rapidity.

Of poor mentality, the patient shows on examination no signs of organic nervous disease.

In this instance no objective evidence points to a local lesion of organic or structural type in the left rolandic area; Spiller's patient, however, was a syphilitic. One of the other cases I have seen was under the care of a colleague who considered exploration justifiable in view of the localised phenomena, and suspected a cortical or subcortical tumour. At the operation (at which I was present) no abnormal cortical condition was found. The interest of these peculiar cases consists in the long duration of the twitching, as though due to persistent stimulation of a cortical reflex arc, and in the absence of any trace of that exhaustion paresis or paralysis which is remarked with some frequency in Jacksonian epilepsy.

Tonic Epilepsy.—Fits characterised by tonic contractions only, to the exclusion of the clonic element, have long been known. Of their semiology and pathogenesis full discussion was given in a paper[17] on decerebrate rigidity published some years ago. It was there argued that many so-called cerebellar fits belong to this tonic group, whose physiological localisation is in posture-effecting mechanisms of mesencephalic site (mainly). The cortical element in an ordinary fit is supposed to be phasic, that is, clonic, although certain investigations (Pike and Elsberg[11]) have demonstrated the occurrence of both clonic and tonic convulsions in decorticate (motor cortex) animals, provided an interval allowing for the return of locomotor reactions elapsed after the initial operation. From the clinical standpoint, fits distinguished by the assumption of tonic attitudes and the occurrence of tonic muscular contractions are most likely to develop in consequence of lesions dissociating physiologically the cerebral hemispheres from the cerebello-mesencephalopontine levels, and the reader is referred to the article mentioned above for a variety of clinico-pathological instances. But brief tonic fits, in some cases not much more than a rather elaborate petit mal seizure, also occur and deserve mention. The following is a recent example.

CASE 4. N. S., female, age 21. When younger, used to have typical myoclonic jerks in the mornings. For the last 18 months, has had fits about once a week. These attacks, repeatedly observed, invariably exhibit the following features: ushering them with a cry, the patient throws her head back and falls backward with a crash; trunk and limb musculatures are seized in rigid tonic contraction, arms flexed, legs extended; cyanosis develops while the tonic contraction is maintained for many seconds. No clonic movement has ever been seen. Relaxation then sets in and consciousness returns.

A few days ago, her head turning as she fell, she broke the bones of her nose and sustained severe ecchymoses of both orbits.

One of her father's brothers was an epileptic lunatic, and another committed suicide. Examination furnishes no evidence of organic subtentorial disease.

In all cases of this kind it is perhaps advisable to suspect an organic and localisable basis for the affection, though doubtless such will not always be discoverable.

Coordinated Epilepsy.—By this term—possibly not the most appropriate that might be devised—the intention is to denote those cases where the movements seen during the attack are coordinated and seemingly purposive, exhibiting objectively to a large extent the features of "voluntary" movements. They may however be aimlessly repeated and in any case do not attain the end to which they may be supposed to be directed.

It is of prime importance to recognise, as I[1] have pointed out elsewhere, that in many epileptic fits quasi-volitional movements occur along with and separate from those called convulsive. "The epileptic patient may, during the fit, make champing movements of the jaws, smack his lips, spit, make clutching movements at the throat; further, when the convulsive phase is over, and while still unconscious, he may make even more elaborate movements of his limbs, such as plucking at his clothes, trying to undress, etc." Most of us must be familiar with post-epileptic confusional states during which all sorts of automatisms of a complex kind are performed without his conscious participation therein. Similar coordinated movement may initiate the attack; I remember a case of left frontal abscess where the patient waved his right arm in circles—as though turning the handle of a barrel-organ—for several seconds ere he fell unconscious and convulsed.

In still other instances the content of the fit is not convulsion but coordination of movement, as in the subjoined case.

Case 5. C. S., male, age 30, has suffered from fits for two years. His attacks occur without warning; he has had one when riding a motor cycle. In them he turns extremely pale and is unconscious of everything, but never falls; he raises his closed fist and blinks with his eyes, then looks sideways as if to see round a corner. The fit is over in about a minute. He never knows that he has had one, till his wife says, "you've just been having one of your funny do's." The nervous system is organically intact.

A maternal uncle suffered from major epilepsy, also a cousin.

As is known, the motor phenomena of petit mal not infrequently are coordinated rather than convulsive or myoclonic.

Thus (1) *before*, *during*, or *after* the phase of actual convulsion or contention of movements individuality of movement may still be preserved; and (2) occasionally only movements of coordination are seen. To me this is a feature of the epileptic seizure that is of profound significance. For it surely helps to break down any artificial semiological barrier between "functional" and other kinds of fit, and indicates differences of degree only, not of quality, between hystero-epilepsy and other epilepsies. Cortical motor centres of the highest level (higher than the rolandic or middle level of Hughlings Jackson) must be implicated if quasi-purposive movements characterise a given fit. Clinically speaking, no arbitrary distinction can be drawn between the elaborate motor phenomena of a hysterical fit and those of the post-epileptic state: both alike represent subconscious or not fully conscious activities released from transcortical inhibition.

These considerations lead to the interesting speculation of the relation of the time factor to the particular guise assumed by epileptic disorders of motility. If the motor elements of a convulsive fit could be extended or lengthened in time, would they not possibly show more coordination than they do? Is not the *swiftness* of the average major fit responsible for the seeming contention of movement, which results in the "single big useless movement," as Jackson termed it, of the tonic stage? What would happen if it were spaced out a little? The clonic movements, "which do nothing but 'mark time,'" exhibit more than a trace of coordination. Were the fit more 'drawn out,' from the standpoint of motion, its features might be so different as to lead to the supposition they were hysterical in nature. The late Sir William Gowers[12] used the apt illustration, in this connexion, of how velocity alters the effect of momentum. "A bullet fired from a rifle makes a round hole in a pane of glass, which it would smash if thrown against it." Variability in length or duration of aura and of post-seizure state of automatism is determined by factors of which we have little or no knowledge, and is accountable for phenomena so diverse as to render individual fits hardly comparable one with another, yet the differences are non-essential. After the briefest of petit mal attacks so rapid as to be scarcely recognisable as such, I have seen a little boy run many yards to hide in any convenient corner—a performance that would have been branded as hysterical had not its real nature been discovered. This epilepsia procursiva, so-called, differs in no fundamental way from a fugue that lasts for hours, days or weeks.

Inhibitory or akinetic Epilepsy.—While the motor syndromes of the epilepsies are ordinarily considered to be essentially kinetic or hyperkinetic, this is in point of fact not universally true. The question arises whether under certain circumstances motionlessness may form a prominent feature of the fit, and it must be answered in the affirmative. Faints and syncopal attacks generally, and some kinds of hysterical fit, are distinguished by relative immobility of the patient; in the case of the former the limb muscles are usually somewhat flaccid and devoid of tone; in the latter less so, since rigidity and motionlessness may occur together. I have had occasion to observe certain attacks which can fairly be taken as coming under the epileptic category, in which similar immobility has been remarked. Years ago both Jackson[13] and Gowers interested themselves in this question; Gowers was of the opinion that discharges in epileptic fits occasionally inhibit, as he found temporary paralysis in some cases after a purely sensory discharge. Jackson referred to cases of epileptiform (Jacksonian) fit in which the patient tells us that his arm "falls dead," there being no spasm in it, whilst the face of the same side is being convulsed. His speculation was that "there may be discharge spreading slowly in a motor centre of the middle level, excessive enough to cause slight after-exhaustion of some of its elements, although one not strong enough to overcome the resistance of lowest motor centres, and thereby to produce actual convulsion."

Brief reference may here be made to a case of unilateral fits which was the subject of prolonged investigation and in which I was able to observe the patient repeatedly during the attacks. Its salient feature was the development of complete flaccid palsy of a temporary character after a sensory Jacksonian aura.

CASE 6. W. C., age 24. Has suffered for six months from attacks of tingling in the left hand and arm, and the left side of the body, occurring every two or three days, followed by weakness of the limbs corresponding.

The fit is preceded by subjective visual phenomena ("bright flashing stars"), without headache, and commences with a strong sensation of tingling in the left fingers and hand; within a few seconds this passes up the left arm, then down the left side of the body rapidly into the left leg, foot, and toes, also appearing in the left face and left half of the tongue. Variable in duration, from a few seconds to a minute or two, it leaves arm, leg and face "dead," and "heavy as lead." When this numbness appears he loses power in his arm and leg—in a second or two this loss is absolutely complete, and the left limbs immobile. The numbness and powerlessness together endure for anything from two to twenty minutes. Recovery of power is rapid and usually simultaneous in arm and leg. Headache usually follows the attack, as a rule bilateral and temporal.

I made many examinations both during and apart from the fits. No definite change in reflexes and no unequivocal sign of organic disease was discovered in the normal periods; in the course of the attack objective loss of sensibility coexisted with flacid palsy, and with exaggeration of the deep reflexes and diminution of the abdominal reflex on the left. In more than one attack patellar and ankle clonus developed, and a slight extensor plantar response.

This striking case (published in greater detail in the article that follows this) is clearly linked to those mentioned by Gowers, and is classifiable as inhibitory epilepsy. Some clinicians might perhaps *call* it a case of migraine, but to do so is merely to change its name; the phenomena themselves are not thereby explained, nor is their epileptiform character thereby invalidated.

The topic, however, has in reality wider limits than may at the first glance be apparent. Attacks of one or other kind in the course of which the patient falls or lies motionless, or becomes incapable for the time of volitional innervation, form a perhaps rather heterogeneous group. Consciousness may or may not be interrupted, slight voluntary movements may be possible, slight involuntary muscular contractions may take place. At this point the question of inhibitory epilepsies merges with the fascinating problems attaching to trance, catalepsy, narcolepsy, and cataplexy. I[14] read a communication on narcolepsy and allied states before the Association of Physicians at their Belfast meeting last June, and this will appear *in extenso* in the next issue of *Brain*: in it a full discussion of these problems, together with a description of various personally observed cases, will be found. Arguments are therein advanced to support the view that some at least of the narcolepsies and epilepsies are interrelated, and that the cataplectic attack, in which the patient sinks to the ground with paralysed and toneless muscles, offers definite resemblances to some of the phenomena commonly called epileptic. This obviates any need to pursue the question further in this article, yet I may take the opportunity of alluding to one or two points.

To illustrate concomitance or sequence of narcoleptic and epileptic symptoms the following case may be cited.

CASE 7. A. G., male, age 21. Had some six epileptic fits between the ages of two and seven years, and three or four more between nine and 14. From the description furnished these would appear to have been characteristic major epilepsy. Since 14, has had no more. Recently however, for some months, has developed a different kind of attack, in which he "loses himself," turns white, may or may not fall, sits or lies still, unable to move, without convulsion, tongue-biting or involuntary sphincter relaxation. These turns are brought on by uproarious laughing; if he laughs less immoderately he gets a "pain at his heart," without the attack following. No signs of organic nervous disease are discoverable.

Here is a clear transition from convulsive epilepsy to inhibitory epilepsy, and I consider the case to be of high significance. The late manifestations are classifiable as cataplectic, the early as epileptic. It is obviously impracticable and unscientific to endeavour to divorce the two from the standpoint of pathogenesis, and the case furnishes us with a good example of how misleading mere nomenclature can become. Another recent case may be reported.

CASE 8. G. P., male, age 52. For the last eighteen months has had what he calls "attacks of trance." The aura is a peculiar smell, always unpleasant and usually likened to that of "burning fat." This olfactory hallucination is of variable duration, and is followed by his becoming "weak all over" and "falling into a trance" in which he turns white, is unable to move, and as a rule is unaware of his surroundings. After four or five minutes he "comes to himself," experiencing at the same time a "peculiar feeling" at the root of his nose, which he has difficulty in describing.

In the instance just given we have the same sequence of temporary generalised immobility after a sensory discharge (in this case of the olfactory order), falling in line with those referred to above. It is evident, I think, that the association constitutes no rarity. At the bidding of intrinsic (and as we shall see immediately, extrinsic) stimuli a process of inhibition, not of excitation, is initiated. This is true of numerous cataplexy cases in the narcoleptic category, and also of occasional cases in the epileptic group. Why one physiological process rather than another should be determined cannot easily be explained, and its discussion would take me too far from the subject at present before us.

II. SENSORY VARIANTS.

1. Reflex epilepsy.
2. Sensory epilepsy.
3. Affective epilepsy.

Reflex Epilepsy.—The term is in usage to signalise cases where an epileptic seizure of one or other kind develops on the heels of an extrinsic sensory excitation of one or other order; formerly known as Brown-Séquard's epilepsy (that experimentalist having produced fits in guineapigs by excitation of cutaneous "epileptogenous" zones after preliminary injury to the cord), it may or may not be associated with spontaneous fits in a given case. Examples of this type are comparatively scarce, although by now a fair number are on

record. In one published by Woodcock[15], that of a little boy, the remark made by his sister was : "Whenever we undress him he has a fit." Undressed on one occasion in the observer's presence, as his stocking was taken off his right leg, he went into an epileptic fit of the classical type, clonic convulsions following tonic, and unconsciousness being complete. I have seen a case in which, when the hat-elastic under a little girl's chin slipped up, hitting the nose, she went off in an epileptic fit. This accident led to the discovery that flicking or tapping the nose always started the attack, which consisted of dilatation of the pupils, tonic spasm of arms and chest, respiratory arrest, cyanosis, and slight frothing at the mouth, lasting in all less than half a minute. (This case was also seen by Dr. Foster Kennedy[16] and is cited by him in a paper alluded to below).

Another variety of reflex epilepsy was termed by Oppenheim acousticomotor epilepsy, and is distinguished by the patient's falling to the ground on hearing a sudden sound. The following is a personally observed example.

Case 9. J. R., female, age 19, has suffered since the age of seven from "falling attacks." She had been knocked down by a taxi two years previously but sustained no actual physical injury. The slightest unexpected noise causes her to collapse to the ground, without unconsciousness, and apparently without any of the usual concomitants of epilepsy, major or minor. So sudden is the attack that she is unable to save herself and has sustained bruises several times. Up to the time of her coming under my observation she had had no epileptic fit. Somatic examination is negative.

A case of this kind is no doubt abortive or incomplete, but it is paralleled and confirmed by one that I have recently been able to see through the kindness of my colleague, Dr. Gordon Holmes. In his case, that of a youth, precisely the same abrupt falling attacks have occurred as an immediate sequel to loud and unexpected sounds, and in addition typical major epileptic seizures have developed. I might also cite another case recorded at some length by Foster Kennedy,[16] in which a boy of 17 would fall immediately to the ground on hearing a sudden noise, not by any means always a loud one. He had suffered from convulsions in infancy, and also had exhibited jerking movements of the left limbs and body when he was younger—seemingly a variety of Jacksonian epilepsy. In this case operation over the parietal cortex revealed an old, organized subdural clot, and was followed by gratifying improvement.

In respect of reflex epilepsy the examples given illustrate the same double process as in the case of the motor variants : an extrinsic stimulus of one or other kind either excites or inhibits. When in consequence of a loud sound a patient falls to the floor it is apparent his posture-maintaining mechanism has undergone inhibition, and the assumption is justifiable that the reaction takes place at mesencephalo-pontine levels. A certain analogy is permissible with the cataplectic seizures of the narcoleptic (*see* above) ; under the influence of stimuli of an emotional kind the limbs give way and the patient sinks to the ground, without loss of consciousness.

Sensory Epilepsy.—That paroxysmal manifestations should be confined to the afferent side is more than a theoretical possibility. We have already

seen how much of a fit may be purely sensory—its aura is a sensation, simple or complex, and in some Jacksonian cases not only does a sensation march along physiological lines (precisely as do the motor symptoms) but it also may represent the whole of the disturbance. An example of sensory epilepsy is the following.

Case 10. M. M., age 43, female. Has had for a period of years recurrent attacks, amounting sometimes to as many as 12-20 a day; at first occurring mainly at the time of the catamenia, they now bear no relation to it. A sudden feeling of faintness, and a slight flush, is succeeded by a strong olfactory hallucination, always pleasant; "it is just as if I had been passing a perfume shop." The lips then become numb, and this is followed by numbness spreading over the right side of the face, right arm, and right leg. There is a certain feeling of stiffness with the numbness, compelling the patient to make voluntary movements of the lips in order to get this feeling away. No true involuntary movement of any kind occurs. The duration of the fit is from three to five minutes. In the course of its development there always appears a faintly conscious background as of voices asking the patient "familiar questions of an ordinary domestic kind."

Careful and repeated examination has not shown that any organic basis for the symptoms exists.

The reader will observe how this case blends with the usual syndrome of uncinate or temporosphenoidal epilepsy; as for its actual clinical type, the manifestations are limited, and confined to the sensory system. Some paroxysmal sensory disorders at higher or lower levels (*e.g.* some of the nemalgias) may well be classifiable with the epilepsies, and the speculation might prove of value as an aid to further observation.

Affective Epilepsy.—We may conveniently include among the sensory variants numerous cases of epilepsy characterised by the fact of their development consecutive to extrinsic or intrinsic affective stimuli. They are to be distinguished from reflex epilepsy in the more technical sense by the definitely affective element of their excitants, though in another sense one might consider the process a "reflex." We should also take note that the variation is not one of epileptic content, but merely of initiation.

"Psychic epilepsy" is a term that has done hard service without ever having been either defined or delimited. "Psychasthenic fits," similarly, have received widely differing connotations (cf. Ernest Jones[17]). It is obvious that expressions of this kind can be applied in psychopathology to fits of a hysterical nature, with which we are not here directly concerned. The reference is rather to ordinary major or minor epileptic fits activated by stimuli of the affective or emotional series. Cases of this sort constitute neither a novelty nor a rarity, and were fully recognised and described long before the war produced them again in some abundance. A typical war case is cited in a paper[2] mentioned above; another case under personal observation may be selected.

Case 11. H. D., male, age 21. Has had typical grand mal attacks for some two years, on an average one every week. A sister, age now 30, has also suffered from typical major attacks for a number of years. One day she fell in a corner of the room in a severe

epileptic seizure; overcome with emotion, and with tears in his eyes, he went to her aid, and as he bent down, with words of endearment on his lips, he suddenly lost consciousness and had an equally severe fit on top of her.

This psychogenic or affective epilepsy is as a fact common enough, and instances need not be duplicated. In comparison with what may be termed unmotivated epilepsy, however, their number is insignificant, and I find myself in radical disagreement with Rows and Bond[13], who assert that in every type of epileptic case a disturbance of consciousness appears, "with which is associated an emotional state," and that "some reaction to express the emotion" can be found in every instance.

III. PSYCHICAL VARIANTS.

The term "psychical equivalents of epilepsy" is objectionable for more than one reason; if any evidence is forthcoming to suggest the same physiological processes of excitation or of inhibition lie behind the psychical symptoms as behind those of epilepsy in the ordinary sense, then the syndromes are not "equivalent" to epilepsy but are as a fact epileptic. Again, we have seen how much of an ordinary fit may consist of phenomena in consciousness (aura, sensory symptoms), belonging therefore to the psychical series, and it is inadvisable to differentiate "psychical" equivalents as though they stood for phenomena never found in the usual epilepsies. Indeed, to harmonise "psycholepsy" with epilepsy is not arduous if we adopt the general physiological principles sketched at the outset. Aldren Turner[19] defines "psychical epileptic equivalents" as "the mental phenomena of the pre- and post-convulsive states, when they occur without convulsion or spasm." In my view this is tantamount to an admission of the *identity*, and not the equivalence, of the phenomena with other epileptic phenomena.

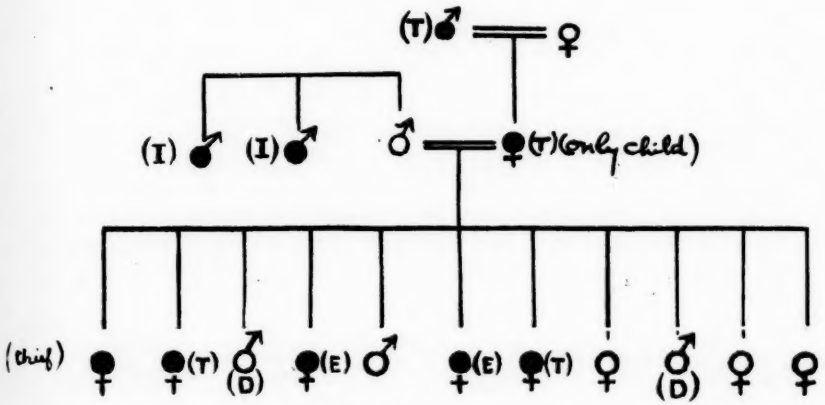
Thus conceived, the phrase must be taken to incorporate diverse symptoms occurring in petit mal, coordinated epilepsy, hystero-epilepsy, post-epileptic stuporose and confusional states, and thereby becomes devoid of any specific usefulness. Including dreamy states in the Jacksonian sense, epileptic mania, fugues and ambulatory automatisms, its perpetuation can serve no scientific or clinical purpose.

I take the chance, nevertheless, of referring briefly to an occasional epileptic concomitant, which has been supposed to form a psychical epileptic variant.

Bad Temper.—The occurrence of instances of bad temper in members of epileptic families is noticed from time to time, and has been subjected to statistical investigation by Davenport.[20] In a number of instances collected by that observer the patient was a sufferer from both epilepsy and bad temper, while in other members of the family they occurred either separately or together. The "epileptic temper" has long been recognised and often described (Ribot,[21] Binswanger,[22] Raecke[23]), and its exhibitions in prodromal and post-paroxysmal periods are familiar to the neurologist; whether substitution of bad temper for epilepsy constitutes the former an epileptic variant is not to be decided by

superficial consideration, since it occurs also in families with insanity as a feature, but without epilepsy. The accompanying diagram gives the details of a family under my observation, in which insanity, epilepsy, and bad temper are prominent traits. The interest resides largely in the appearance of bad temper in three members, none of whom has ever had an epileptic fit; but as insanity also is a familiar inheritance it rather complicates the issue.

FIG. I.



I = Insanity.
T = Temper.
E = Epilepsy.
D = Died in infancy.

Davenport's conclusion is to the effect that since violent temper can be shown to occur familially without either epilepsy or insanity it should be regarded not as an "equivalent" of epilepsy but as due to a factor which has its greatest effect when acting on a nervous system, especially liable to the other.

IV. VISCERAL VARIANTS.

Obscurities arise when, finally, we approach the question of epileptic syndromes of viscerosensory and visceromotor origin. Here we must proceed warily, content to observe if we cannot yet explain. On general grounds, participation of visceral centres in epileptic manifestations must of course be conceded. The sudden pallor of many cases of petit mal, not to mention those of "nervous faints," the sphincter relaxation of minor or major fits, are instances in point. As the "discharging lesion" sweeps over different levels it would be curious to find selectivity exercised; the discharge is "brutal," as Jackson said, and does not spare mechanisms belonging to the sympathetic system, in their central connexions. What interests us for the moment, however, is the possibility of epileptic variants the outcome of physiological disorder localised largely if not entirely in sympathetic centres,

to the exclusion of sensorimotor phenomena of somatic type. One variety can be clearly differentiated; others, more fragmentary, can also be exemplified.

Vasovagal Attacks.—This name, which we owe to Gowers[12] (1907), has never received the sanction of common usage: "nerve-storms," "pseudo-angina" have a certain vogue, and "anxiety neurosis" a greater one, yet for a number of reasons I prefer that of vagal or vasovagal attack. For years it has been my custom to collect cases falling under this heading, and I[24] have drawn attention to them on several occasions, but the idea is not one, as far as I can gauge, that has taken its place among the conceptions of practice. The expression "anxiety neurosis," as an inclusive term, is misleading, since it is based on the view that the manifestations, both mental and physical, which here concern us, are "the inevitable accompaniment of conflict within the personality" (Gordon[25]). To this assumption I cannot subscribe. That the phenomena do in fact often enough thus arise I fully admit, but my contention is that in other circumstances they are adequately described as epileptic variants (in still others, they follow from actual visceral disease). The error has its origin in ignoring the well-established general principle that a given clinical syndrome may be of diverse etiology and of differing physiological localisation.

Speaking generally, the symptoms of the vasovagal attack are referable to transient disturbance of function mainly in the organs supplied by the pneumogastric nerve, and since implication of the motor vagus is doubtful the probability is they develop from disorder of the dorsal vagal nucleus in the medulla and of the vasomotor centre in juxtaposition. As a result, the patient complains of attacks of palpitation at the heart, difficulty in breathing, a suffocating feeling in the throat, hot flushes, icy coldness, sweating, shivering and trembling, nausea and other epigastric sensations, and often, also, an indescribably distressing sensation of impending trouble, of fear, even of imminent dissolution. One or more of these symptoms, doubtless, may occur as an episodic syndrome in the course of either functional or organic nervous disease, but in the general form already outlined they are periodic and but slightly varying, and their due recognition is a matter of clinical importance. I shall select one or two illustrations out of a large number.

CASE 12. N. J., female, age 28. Has suffered for nine months from attacks which are exemplified in the following excerpts from a description with which she furnished me at my request.

"I was sitting in the garden with another patient at the time and had a quiet feeling as though I did not wish to speak or take notice of anybody or anything, but seemed to be living inside myself. At the same time everything impressed itself upon my mind to an unnaturally forcible extent. I tried to take no notice but to rouse myself and think of something pleasant but could not do so as my thoughts seemed confined; even familiar objects around me, and the birds singing, seemed different and more intense.

"All the time I was conscious of a dread that something was going to happen, accompanied by a fear of death. I had a feeling of slight tingling or electricity all through me and was exhausted, cold and clammy, as though all the blood and strength had gone out of me. Then my heart began to beat rather heavily and quickly, which affected my breathing, making me take my breath in gasps. After that I started shaking all over and then a warm feeling came down over me, like the blood returning, especially in my

head and face, and tears came involuntarily. My feet remained cold all the time. When it had passed off I felt better in my mind but weak and shaky in my body, and depressed."

By way of amplification it may be stated that nausea and a peculiar feeling in the stomach were also noted towards the outset, and that the flush which developed as the attack subsided was usually accompanied by some perspiration.

A cousin of the patient suffered from major epilepsy. Examination of all the bodily somatic systems has been consistently negative.

Case 13. C. C., female, age 25. For several months has complained of attacks of "the nerves." On investigation these turn out to consist of palpitation at the heart, difficulty in breathing, sweating, icy-cold extremities, and generalised trembling, together with an anguished feeling of impending death. The attacks vary in duration, from a quarter of an hour to as long as four hours, and after every one there is urgent and almost involuntary defæcation.

Family history and objective examination are alike negative.

Case 14. F. D., male, age 39, a member of the medical profession. Has suffered from typica major epileptic seizures ever since his student days. These fits are severe, and are characterised by cyanosis, convulsions, tongue-biting, incontinence, etc. The aura (not constant) consists of a peculiar repetition in the left ear of a word or series of words (not always the same, and not always clearly recollected).

In addition, the patient has frequent "sensations." These also are ushered in by a well-defined repetition of words in the left ear, and consist of a sharp feeling at the heart, "something like a tug," a sense of great difficulty in getting breath, icy-cold extremities, intense fear (apparently of developing a fit); then their place is taken by flushing of neck and face, the heart beats more strongly and quickly, giving a sense of relief, the limbs shake, and pronounced upward eructation of wind always ensues.

There are no signs of organic nervous disease and no structural visceral changes.

Case 15. I. H., female, age 10. Has had "fits" for the last six weeks. These commence by a sinking feeling in the stomach, associated with giddiness and a form of micropsia; "everything looks quite tiny." Then come severe palpitation, icy coldness and deathly pallor. "I feel as if I were going to die, my heart keeps on throbbing so." Bad headache, cold shivers, and numbness of the right arm and right side of the face round off the attack, which lasts about a quarter of an hour.

On examination no organic nervous disease and no visceral cause for the symptoms were discovered.

This patient came again to hospital eight years later (age 18) with a history that the attacks had ceased for a period of several years. In their place another type now occurred, in which the aura was "blindness" and which from its characters was suggestive of migraine.

The above cases are chosen from a large number because they illustrate various points of significance. They may be amplified generally by reference in a little more detail to the symptoms as they unfold themselves *seriatim*.

(1) The gastric or epigastric sensation is often described as though that organ were moving or turning over, associated with a sick feeling, or as though it were empty or void. "It is a creepy feeling, as if the stomach moves about"; "like being on the waves of the sea"; "as if there were a hollow in the stomach, or a hole"; "as if I were hungry and yet I don't feel hungry."

(2) The cardiac sensation is highly variable, but actual praecordial or cardiac pain is unquestionably rare. Rather the patient notes fluttering, racing, thudding, thumping, of the organ; cardiac arrest is a seeming actuality. In one case, cardiac pain with radiation down the left arm took place.

(3) Respiratory phenomena consist of tachypnoea, less often of bradypnoea, and of unpleasant subjective sensations such as a choking, suffocating gulping feeling: "my pulse comes up into my throat and chokes me."

(4) Concomitant vasomotor symptoms, equally variable, consist in a coldness like marble—"the coldness of death"—with shivering, trembling, shaking; a cold and clammy perspiration breaks out. Warm or hot feelings suffuse the limbs afterwards. "I feel as if all the blood had gone out of me and that I was left a stone; then I begin to glow all over."

(5) It is of great interest to note that in not a few cases the patient voids large quantities of limpid urine after the attack: in one of the instances cited above defaecation occurred.

(6) As for accompanying phenomena of the psychical series, we note the sensation of *angoisse*, of impending catastrophe, of imminent death. Words of the patients are far more graphic than a laboured description: "Oh, I'm going! I'm going!" "I think it's a death struggle and I say my prayers every time." "I feel, is this it? Is this death at last?" In other instances the feeling is brief and indefinite, yet usually with a sinister content. While fear of one or other degree is common, I have not come across any cases where a conscious cause for it existed in the patient's mind.

We also note that there is no loss of consciousness, though frequently a sense of unreality, "as in a dream," closely allied to the "dreamy state" of uncinate epilepsy. Sometimes the senses are preternaturally acute, on the other hand; the patient feels abnormally on the alert. Again, particular attention is drawn to the fact that on occasion he is conscious of a sensation of "being unable to move," as if in a trance. "I struggle inwardly, but nothing happens. I try to call out, but I say nothing. I feel if only I could move all would come right." "I hear everything they say to me but I cannot utter a single sound."

This latter circumstance links the condition in some instances to what I[14] have shown is characteristic of numerous narcoleptic and cataplectic fits—viz., inability to move or to innervate, and constitutes a phenomenon of inhibition accompanying those of release, as in some other epileptic variants.

These vasovagal fits frequently arise in cases with a family history of epilepsy, migraine, or insanity. Thus one of my patients suffered from characteristic attacks for a number of years: his mother's brother was insane, and two of his father's brothers had epilepsy; his mother, and a brother, were both martyrs to migraine. Sometimes no evidence of neuropathic inheritance is forthcoming, as also in many epileptic individuals. I am not concerned here with the appearance of anxiety neurosis as a sequal to "conflict within the personality," hackneyed though such a development is thought to be. Careful investigation fails to reveal any trace of "conflict" in the cases narrated above: and the "coitus interruptus" theory is of course inapplicable, not merely in the case of juveniles, but also in others in which the practice has never been indulged. To my mind the theory that vasovagal phenomena can in many

instances be considered epileptic variants does no violence to the facts and is consonant with what we know of epileptic manifestations otherwise. I do not believe we should expect the reactions of visceral centres in the neuraxis to be identical with those of somatic centres; allowance must be made for differences, histological and physiological, in the ganglionic collections that are the seat of "discharging lesions." The "extended" nature of the vasovagal attack cannot of itself form an insuperable obstacle in the way of acceptance of the hypothesis. If a faint is a bulbar inhibitory fit, a vasovagal attack can be a bulbar fit of a less rapid, less "brutal," more coordinated kind.

Precaution is desirable, on the other hand, before a given case of this bulbar syndrome can be entered as epileptic. Under very differing etiological circumstances phenomena resembling those that have been sketched can make their appearance. Shock, anaphylaxis, disorders of intracranial circulation, commotio cerebralis, and so forth, are pathological conditions in the course of which bulbar symptoms can and do arise. Attention was directed by Lévy[28] long ago to the ease with which bulbar disarray (*affolement bulbaire*) develops episodically, while in recent years among the sequelæ of epidemic encephalitis have occurred complex types of respiratory disturbance only a minority of which are classifiable with lowest level (ponto-bulbar) fits, though few if any are strictly comparable with the vasovagal attack.

Other Visceral Fits.—Other types of recurring seizure in which symptoms referable to central visceral disorder make their appearance have come under notice from time to time, and some are rather difficult to classify, as the subjoined examples prove.

Case 16. C. S., male, age 11. A very intelligent boy, and a scholarship winner, he has had a series of attacks during the last 12 months which are heralded by deep and incessant yawning, for perhaps half an hour. He turns deadly pale and then starts to vomit, while violent frontal headache develops. The whole thing may last for rather less than an hour.

Objective examinations has repeatedly proved negative. There is no family history of epilepsy, migraine or other analogous condition.

Case 17. C. L., male, age 25. Has had peculiar fits for about one year, recurring on an average at fortnightly intervals. They are constituted by continual yawning, with constant voiding of urine, and are followed by violent giddiness, headache, and occasional vomiting.

There is a family history of epilepsy. No organic signs have been found on repeated examination.

The explanation of these cases is not simple. One might hazard the speculation that they represent some disturbance of third ventricle visceral centres concerned in the regulation of sleep and of water-interchange, with irradiation to visceral centres situated in the floor of the fourth ventricle.

As clinicians, we must be prepared to meet with syndromes, such as these, which do not fit readily into hard-and-fast schemata. Not enough is known of the normal functions of the sympathetic system to enable detection of its disorders to be recognised with precision. We may only suspect certain visceral syndromes to be the outcome of functional disarrangement of

abdominal plexuses ; we still are largely in the dark as regards those derived from changes at higher visceral levels. It is our task, therefore, with open mind to observe symptoms, collect data, and collate instances, and imagination may supply the light whereby we shall see eventually how heterogeneous syndromes slip into their place in the physiological puzzle.

REFERENCES.

[1] Wilson, Kinnier, *Brit. Med. Jour.*, 1926, ii, 1.
[2] Wilson, Kinnier, *Brit. Med. Jour.*, 1927, i, 221.
[3] Muskens, L. J. J., *Epilepsia*, 1909, i, 161. Also *Die Epilepsie*, Berlin, 1926.
[4] Crouzen, O., and Bouttier, *Soc. méd. des Hôp.*, 1921, xlv, 278.
[5] Koshewnikow, A., *Neurol. Centralb.*, 1895, xiv, 47.
[6] Orlowski, S., *Neurol. Centralb.*, 1896, xv, 526.
[7] Choroschko, *Neurol. Centralb.*, 1908, xxvii, 279.
[8] Spiller, W. G., *Jour. Amer. Med. Assoc.*, 1909, lii, 1921.
[9] Bruns, B., cited by Spiller (Ref. 8).
[10] Wilson, Kinnier, *Brain*, 1920, xliii, 220.
[11] Pike, F. H., and Elsberg, C. A., *Amer. Jour. Physiol.*, 1925, lxxii, 337.
[12] Gowers, W., *The Borderland of Epilepsy*, London, 1907.
[13] Jackson, Hughlings, *Brit. Med. Jour.*, 1890, i.
[14] Wilson, Kinnier, *Brain*, 1928.
[15] Woodcock, H., *Lancet*, 1919, ii, 60.
[16] Kennedy, Foster, *Arch. of Neurol. and Psychiat.*, 1923, ix, 567.
[17] Jones, Ernest, *Jour. Abnorm. Psychol.*, 1907, ii, 218.
[18] Rows, R. G., and Bond, W. E., *Epilepsy*, London, 1926.
[19] Turner, W. Aldren, *Epilepsy*, London, 1907.
[20] Davenport, C. B., *Jour. Nerv. Ment. Dis.*, 1915, xlii, 593.
[21] Ribot, T., *The Psychology of the Emotions*, New York, 1896.
[22] Binswanger, O., *Die Epilepsie*, Vienna, 1899.
[23] Raecke, *Transitorische Bewusstseinstörungen der Epileptiker*, Halle, 1903.
[24] Wilson, Kinnier, *Brit. Med. Jour.*, 1913, i, 1257. Also *Lancet*, 1913, ii, 1677.
[25] Gordon, R. G., *The Neurotic Personality*, London, 1927.
[26] Lévi, L., *Presse méd.*, 1905, xiii, 433.

Short Notes and Clinical Cases.

A CASE OF EPILEPSY ILLUSTRATING THE LOSS OF CORTICAL CONTROL OVER LOWER FUNCTIONS.

By R. G. GORDON, Bath.

Since Hughlings Jackson pointed out that a negative lesion could not be held responsible for positive symptoms, the phenomena of epileptiform convulsions have gradually come to be regarded as the activities of lower levels in the nervous hierarchy released from control of the higher levels. It is seldom, however, that we can actually observe behaviour on the part of the patient which directly supports the truth of this hypothesis. As a general rule the preliminary phases of an epileptic fit are so unexpected and are passed through so rapidly that we are quite unable to detect any gradual abrogation of higher control before the familiar phenomena of the actual fit occur. For this reason it seems worth while to record the observations described below, as although they only represent what happened on one occasion and it has unfortunately been impossible to repeat the record then obtained they do seem to afford that direct proof of Jackson's hypothesis which is so difficult to obtain.

R. P. was a boy of 12 years who was brought to the O.P. Department with the complaint of epileptic fits. His mother gave a history of natural labour with this child, who was the only one of the family. No relevant family history was obtained as to epilepsy or other nervous or mental trouble. The patient had a convulsion lasting one hour when eleven months old and this was said to be due to teething. Otherwise his development was normal until he reached the age of six and a half, when he was said to have had a severe convulsion or series of convulsions lasting from four to five hours and affecting both sides of his body. It was noticed that the last movements to pass off were on the left side of his face. At the age of seven he fell off a shed on to his head and was reported to have been unconscious for a minute or two, but he had no convulsion and no doctor was called in. At the age of nine he was seen by his own doctor for attacks of what was described as petit mal. It was noted at this time that the patient was a very nervous child, suffering from night terrors. In March 1923, when aged ten, he had a series of epileptiform convulsions which were characterised by deviation of eyes to the right; it was noted that the fits started on the right side of the body and lasted for an hour and a half. Since this time he has frequently had

attacks of petit mal in which he " goes white round the mouth, is unconscious and speaks incoherently." In October 1924 he had a series of grand mal attacks followed by left-sided headaches. In June 1925 while at school he had the fit which is the subject of the present communication. On examination shortly after this he was found to be suffering from enlarged tonsils and adenoids, but he was otherwise physically fit. No reflex changes were discovered; he was mentally bright but was obviously a nervous child and apparently suffered continuously from night terrors. The parents were over-anxious and were always worrying about the boy's health.

On the occasion referred to above, in June 1925, he was at school and engaged in writing to dictation. Words were read out as recorded in the left-hand column. His replies were correct as far as the word " quarrying." After this his replies are recorded in the right-hand column. At the point "-ation " he fell over unconscious and had a severe epileptiform convulsion.

Pillaged orient	
Oriental corrupted	
Destined desfination	
Elaborate projecting	
Minaret delicately	
12 days, 2 hrs., 50, 18 mins.	
Bazaar adopted horizon	
System cylinder cylindrical	
Occasion occasionally	
quarry quarrying	
Precisely revolution	3% Ronald rewpla
Success successive	cing sucppess sucpessing
Anarchy anarchist	another anerihind
Opium associate .	nownoun where
Association episode.	assovere assovers-
	ation.

The above is an exact copy of his exercise book.

As has been said this seems to illustrate very prettily the true nature of an epileptiform fit as a release from higher cortical control. The jargon agraphia is a clear manifestation of the loss of cortical control culminating in a complete liberation of the lower motor functions which manifest themselves as a convulsion.

Editorial.

A SUGGESTED ANALOGY BETWEEN PSYCHOLOGY AND NEUROLOGY.

THE relation between medical psychology and neurology has been, although relatively affable, a tangential one.

This fact, however regrettable from the point of view of science as a whole, is determined by the nature of the respective fields of work. Each field is a more or less closed system with its own basic conceptions and technical methods, and nothing but confusion would result from an attempt to superimpose the concepts of the one upon the methods of the other. But although it is not in the nature of parallel lines to meet, their parallelism is in no sense violated by the formation of anastomoses, by which contact and kinship can be maintained. The anastomosis, if it be a real one and not merely a politic adjustment, will derive its effectiveness from the appreciation of an analogy that exists, either in the actual material, or in the momentary situation between the two systems.

Between modern therapeutic psychology and neurology a very striking and material analogy is actually present. Neurology has to deal with two distinct and essentially contrasting neural systems—the cerebrospinal and the sympathetic. The former is a later and more differentiated morphological development, while the latter has retained the pristine, vegetative character belonging to an infinitely older and more archaic pattern. No system in the body has been so exhaustively explored—both functionally and structurally—as the one: whereas with respect to the other neurology is still groping in a somewhat vague *clair-obscur*.

This situation finds an exact parallel in the sphere of psychology, where two contrasting mental systems or modes—the conscious and the unconscious—have to be taken into account, and where the concepts and methodology appropriate to the one system have proved inapplicable to the other. The clear-cut formulations and distinct categories of the academic psychologists, though lacking nothing in crystalline conceptual

clarity, fail—and for this very reason—to embrace the irrational dynamic character of the unconscious mental processes. Western scientific consciousness is wedded to the principle of direct or rational causality, and every system, as for example the cerebrospinal where this principle can be exhaustively followed and demonstrated, offers a clear field for its operation. But there are systems, as there are certain aspects of reality, which do not correspond to this principle; systems where the causal nexus has oblique, indirect, or irrational connexions, so that the attempt to apply the direct, causal interpretation only serves to entangle, and not to clarify, our thought.

The unconscious is such a system—if one may designate this ambiguous and chaotic terrain with such an orderly term—and the task of psychology has been to formulate concepts that accord with its irrational, dynamic character. Naturally the concept, if valid at all, must also partake of the nature of the thing that it embraces. But nothing is so vigorously excluded by the intellect as the irrational elements of life. Hence the intuitive concepts, which the very nature of the unconscious mental processes force us to adopt, are liable to cause the liveliest resistance to a purely rational mind; and similarly with the technique. The entirely irrational character of the dream, and of the free associations used in the elaboration of dream-material, is a constant perplexity to the mind that is ruled exclusively by the *ratio*.

Since the intellect unaided is unable to construct a conceptual framework that can embrace every aspect of reality, clearly another function, whose nature is essentially irrational, must be brought in to collaborate with the intellect. This function is found in intuition, and the fundamental restatement of biological conceptions by Haldane, and of psychological conceptions by Jung has already demonstrated the immense value of introducing this new dimension of the mind into our scientific hypotheses. It could probably be shown that every original investigator has made constant use of his intuition as a 'path-finder,' but it could also be shown that scarcely one of the scientific leaders of last century gave sufficient credit to his intuition to give it equal place with the intellect in the formulation of a general hypothesis.

The nature of intuition is synthetic; it perceives analogies, parallels, conjunctions and associations; it links and joins and overleaps. But above all it does not run in blinkers along the firm highway of direct causality. It tries experimental by-paths, and even leaps hedges with winged freedom. The

rational intellect has a strong integrity, and it is this loose freedom and irregularity of method by which intuition achieves its goal, that seem to undermine the feeling of intellectual integrity. Moreover, the intellect has good reason to suspect intuition—how often in its history has not science been seduced along the by-paths of superstition through its nefarious influence? But superstition results from uncriticized intuition.

Because of its pristine, pre-rational character intuition always speaks in the primitive language of metaphor. An intuition is a concretistic 'as though' statement, and therefore always needs to be transposed into more abstract terms. The transposing of the intuitive metaphor into organized mental forms, and the testing of the intuitive findings in the world of objective reality is the work of the intellect. Thus these two apparently contradictory functions can actually form an excellent partnership, just in so far as either function respects the other's scope and character.

Direct causality is therefore not an absolutely final interpretation. It is a category of the reason, as Kant showed. Another way of viewing phenomena is by the way of synchronicity, which is the way of intuition. Things happening together, things related, as it were, transversely rather than sequentially, this is the irrational dimension of the unconscious which intuition apprehends. For we must remember that the unconscious comprises all the other aspects of the world and reality that escape conscious recognition, and that intuition perceives by way of the unconscious. Hence it is that intuition is particularly well fitted to inform us concerning just those factors which normally escape the observation, that is controlled exclusively by the reason.

It is useless to seek analogies where none exist, but is there not in fact a suggestive parallel between the contrasting psychic systems, viz., the conscious and the unconscious, with their antithetic principles of orientation, and the contrasting neural systems, viz., the cerebrospinal and sympathetic, with their distinct, even opposite, modes of functioning? Is it not more than mere coincidence perhaps that the cerebrospinal system seems to invite the explanatory principle of direct or sequential causality, while sympathetic symptomatology invariably presents a picture of synchronous events and modifications, the interrelations of which are often quite comprehensible, although their causality may be, and usually is, obscure? This analogy is so striking that the view has been suggested that the unconscious, which undoubtedly displays

clarity, fail—and for this very reason—to embrace the irrational dynamic character of the unconscious mental processes. Western scientific consciousness is wedded to the principle of direct or rational causality, and every system, as for example the cerebrospinal where this principle can be exhaustively followed and demonstrated, offers a clear field for its operation. But there are systems, as there are certain aspects of reality, which do not correspond to this principle; systems where the causal nexus has oblique, indirect, or irrational connexions, so that the attempt to apply the direct, causal interpretation only serves to entangle, and not to clarify, our thought.

The unconscious is such a system—if one may designate this ambiguous and chaotic terrain with such an orderly term—and the task of psychology has been to formulate concepts that accord with its irrational, dynamic character. Naturally the concept, if valid at all, must also partake of the nature of the thing that it embraces. But nothing is so vigorously excluded by the intellect as the irrational elements of life. Hence the intuitive concepts, which the very nature of the unconscious mental processes force us to adopt, are liable to cause the liveliest resistance to a purely rational mind; and similarly with the technique. The entirely irrational character of the dream, and of the free associations used in the elaboration of dream-material, is a constant perplexity to the mind that is ruled exclusively by the *ratio*.

Since the intellect unaided is unable to construct a conceptual framework that can embrace every aspect of reality, clearly another function, whose nature is essentially irrational, must be brought in to collaborate with the intellect. This function is found in intuition, and the fundamental restatement of biological conceptions by Haldane, and of psychological conceptions by Jung has already demonstrated the immense value of introducing this new dimension of the mind into our scientific hypotheses. It could probably be shown that every original investigator has made constant use of his intuition as a 'path-finder,' but it could also be shown that scarcely one of the scientific leaders of last century gave sufficient credit to his intuition to give it equal place with the intellect in the formulation of a general hypothesis.

The nature of intuition is synthetic; it perceives analogies, parallels, conjunctions and associations; it links and joins and overleaps. But above all it does not run in blinkers along the firm highway of direct causality. It tries experimental by-paths, and even leaps hedges with winged freedom. The

rational intellect has a strong integrity, and it is this loose freedom and irregularity of method by which intuition achieves its goal, that seem to undermine the feeling of intellectual integrity. Moreover, the intellect has good reason to suspect intuition—how often in its history has not science been seduced along the by-paths of superstition through its nefarious influence? But superstition results from uncriticized intuition.

Because of its pristine, pre-rational character intuition always speaks in the primitive language of metaphor. An intuition is a concretistic 'as though' statement, and therefore always needs to be transposed into more abstract terms. The transposing of the intuitive metaphor into organized mental forms, and the testing of the intuitive findings in the world of objective reality is the work of the intellect. Thus these two apparently contradictory functions can actually form an excellent partnership, just in so far as either function respects the other's scope and character.

Direct causality is therefore not an absolutely final interpretation. It is a category of the reason, as Kant showed. Another way of viewing phenomena is by the way of synchronicity, which is the way of intuition. Things happening together, things related, as it were, transversely rather than sequentially, this is the irrational dimension of the unconscious which intuition apprehends. For we must remember that the unconscious comprises all the other aspects of the world and reality that escape conscious recognition, and that intuition perceives by way of the unconscious. Hence it is that intuition is particularly well fitted to inform us concerning just those factors which normally escape the observation, that is controlled exclusively by the reason.

It is useless to seek analogies where none exist, but is there not in fact a suggestive parallel between the contrasting psychic systems, viz., the conscious and the unconscious, with their antithetic principles of orientation, and the contrasting neural systems, viz., the cerebrospinal and sympathetic, with their distinct, even opposite, modes of functioning? Is it not more than mere coincidence perhaps that the cerebrospinal system seems to invite the explanatory principle of direct or sequential causality, while sympathetic symptomatology invariably presents a picture of synchronous events and modifications, the interrelations of which are often quite comprehensible, although their causality may be, and usually is, obscure? This analogy is so striking that the view has been suggested that the unconscious, which undoubtedly displays

an archaic mode of functioning, is actually situated in the sympathetic system, just as it is assumed that the conscious psyche is situated in the brain. If this analogy is felt to have substance by the neurologists, the task of restating our fundamental conceptions may not fall upon psychology alone, but the intuitive or synthetic point of view will also become a necessity for the handling of disorders of the sympathetic system that at present escape our comprehension.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[123] **The anatomy of the temporal operculum** (Zur Anatomie der operkularen Temporalregion).—KAKESHITA. *Arb. a. d. Wien. neurol. Institut.*, 1926, xxvii, 292.

THIS study consists of a thorough histological examination of the temporal operculum and a macroscopic and microscopic comparison of corresponding areas of the two sides of the brain. The upper surface of the temporal lobe can be divided primarily into three regions, the temporo-polar region, the gyrus temporalis magnus, and, posteriorly, the region of the smaller transverse gyri. The temporo-polar region is divided by more or less distinct markings into three sub-regions which are called anterior, middle and posterior. The gyrus temporalis magnus includes the gyrus transversus magnus and, on its medial side, the para-insular area which in some brains appears as a gyrus, in others as a depression. The transverse temporal gyri behind are usually three in number and may be as many as five ; they are not distinctly demarcated from the transitional area posterior to them.

In nearly all the ten cases examined this region of the brain showed definitely greater macroscopic development on the left side than on the right. Not only had it more numerous and deeper markings, but the region as a whole had often greater length, the difference in one case being as much as three centimetres ($9\frac{1}{2} : 6\frac{1}{2}$).

According to the cyto-architecture seven areas are described in the region studied—the area acoustica, the areae paracousticæ immediately anterior and posterior to it, an anterior polar area, a posterior polar area, the area parinsularis adjoining the insula, and an area agranularis in the transition zone of the transverse gyri posteriorly ; it is doubtful whether this last is to be included as part of the operculum. In the five cases histologically compared great differences were found in the sizes of these areas on the two sides of the brain, and also on the same side from case to case. Generally the extent of the area paracoustica posterior was about the same on the two sides. In the first case the area acoustica was very much larger on the left than on the right and the anterior paracoustic area somewhat larger too, and there was no cortical region on the left showing the structure of the area polaris posterior. In the second and third cases the left and right sides were approximately the same. In the fourth case the area paracoustica anterior was large on the left but hardly existed at all on the right, the variation being again at the expense of the area

polaris posterior. In the fifth case the area acoustica on the left was enormously larger than on the right, but the left paracoustic area was somewhat smaller than the right ; there was no area polaris posterior on either side.

These cases indicate that the area acoustica is frequently of much greater extent on the left side, and that the combined extent of the area acoustica and the area paracoustica anterior is usually greater on the left. A most important fact is that the extent of the acoustic area differs widely from case to case and is not by any means always limited to the gyrus transversus magnus. Further, there is practically no correspondence between the extent of the various structural cortical areas described and the superficial markings of the region.

J. P. M.

[124] **The corticothalamic paths in certain small mammals** (Les voies corticothalamiques chez quelques petits mammifères).—D'Hollander and Ghisoland. *Jour. de neurol. et de psychiat.*, 1927, xxvii, 497.

In this article only a small portion of the corticothalamic paths is described, namely, their terminal portion in the thalamus. The authors are not concerned to show that the fibres in question have a cortical origin : that has been demonstrated in a previous paper ; their object is to describe the mode of termination of the fibres and to compare it in different species of animals.

They show that in the rat and hedgehog the corticothalamic fibres break up into well-defined groups which enter certain constant nuclear divisions. To the anterior group of thalamic nuclei—described as antero-external, antero-internal, internal paratænial and external paratænial—individual bundles of fibres pass from the internal capsule under the floor of the lateral ventricle, and turn upwards in the thalamus. More posteriorly in the thalamus fibres in considerable numbers are shown passing to the ventral, medial and lateral nuclei ; those to the central and medial enter transversely from the internal capsule, but those to the lateral come via the ventral part of the capsule, curve round the inner border of the zona reticulata and pass almost vertically dorsalwards to the lateral nucleus. Still more posteriorly groups of fibres are shown entering the posterior nucleus in a similar manner, curving upwards through the zona reticulata. Other fibres pass superficially on the dorsal surface of the thalamus and enter the pre-tectal area by passing over and through the geniculate bodies. To the corpora quadrigemina cortical fibres pass in two divisions, a superficial division which passes down through the internal capsule to the peduncle and then turns dorsally and reaches the tectum by a superficial path over and through the external geniculate body, and a deep division which leaves the internal capsule and passes through the zona reticulata and thence dorsalwards.

There are certain relative variations in the size of these groups in the rat and hedgehog. The grouping in the brain of the mouse corresponds closely to that in the rat, and the grouping in the brain of the guineapig is almost a replica of that in the hedgehog.

The relative sizes of these fibre groups in the animals described supports the views of Kappers regarding the functions of certain of the thalamic nuclei—that the anterior group is chiefly associated with smell, the medial nucleus with the sensory system of the trigeminal, and the posterior nucleus with sight.

J. P. M.

[125] **The bloodvessel distribution of the cerebral cortex** (Ein Beitrag zur Kenntnis der Gefässverteilung in der Hirnrinde).—R. LORENTE DE NÒ. *Jour. f. Psychol. u. Neurol.*, 1927, xxxv, 19.

USING the technique of Golgi-Cox, the author has made a fine series of sections of the cortex to show the capillary and small bloodvessel distribution, and these are reproduced in a number of beautiful plates. He concludes that there can be no regular and constant following of a particular route in the capillary network by blood from a given source, and also that the capillaries must be ever widening and narrowing according to activity or otherwise of cell-groups. He thinks it possible this mechanism is activated by the fibres of glia-cells attached to capillary walls.

S. A. K. W.

[126] **Cerebral circulation.**—H. S. HOWE and E. MCKINLEY. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 81.

AN account of the study of the cerebral circulation in certain lower animals by direct microscopic observation. Mechanical stimulation was without the slightest effect on the calibre of the arteries, veins or capillaries, but faradic stimulation of arterioles caused a sharp contraction at the point stimulated. Some of the capillaries showed similar responses, but the veins were unaffected. Direct application of epinephrine in strengths varying from 1:1000 to 1:10,000 caused dilatation in the cerebral vessels of the frog, but was usually without effect in the cat or rabbit.

Intravenous injections of various substances such as epinephrine, pituitary extract, paraldehyde, hypertonic salt solution and India ink affected the rate of circulation in different ways but for the most part no perceptible change in the size of any of the cerebral vessels was encountered. R. M. S.

[127] **Experimental variation of the secretion of cerebrospinal fluid** (Ueber experimentelle Beeinflussung der Liquorsecretion).—HIRSCH-TABOR and VOLLMAR. *Münch. med. Woch.*, 1927, lxxiv, 709.

THE findings of Dixon and Halliburton regarding the effect of injection of certain substances into the blood stream on the secretion of cerebrospinal fluid (in dogs and cats) do not, except in small measure, receive confirmation from the experiments of Hirsch-Tabor and Vollmar. Injection of extracts of brain substance or of choroid plexus in most cases failed to produce any increase in the amount of fluid secreted. Healthy human fluid and serum seemed to have

no effect, but fluids in which the Wassermann reaction was positive caused an increase of the flow. This finding may depend on the presence of break-down products of the brain in the syphilitic fluid.

Clinical adaptation of some of this work is foreshadowed. J. P. M.

[128] **Intraocular and intracranial pressure.**—F. Fremont Smith and H. S. Forbes. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 550.

The burden of evidence of many workers suggests that the intraocular and cerebrospinal fluids are of the nature of dialyzates in simple membrane equilibrium with the plasma. The rate of dialysis depends on the balance between hydrostatic capillary pressure and the osmotic pressure of the plasma proteins; so that given a constant composition of plasma the filtration rate will vary directly with the capillary blood pressure.

Ludwig, and later, Leonard Hill and others have shown that capillary pressure depends far more on venous than on arterial blood pressure, and hence both the formation and absorption rates of cerebrospinal fluid and of the aqueous humour are governed by the venous blood pressure. In an elaborate series of experiments these arguments are tested and the authors conclude that although the intraocular and intracranial pressures are not directly interdependent, changes in hydrostatic or osmotic blood pressure produce parallel pressure changes in the eye and in the cranium.

C. J. E. C.

NEUROPATHOLOGY.

[129] **New methods for impregnation of microglia** (Nuovi metodi per l'impregnazione della microglia).—D. Bolsi. *Giorn. d. R. Accad. Med. Torino*, 1927, xc, 262.

After referring to methods already in use, the author offers the following technique of his own as giving excellent results:

1. Fix pieces in pyridine 5 c.c., acetone 5 c.c., formol 15 c.c., ammonium bromide 3 gr., distilled water 75 c.c.

2. Cut frozen sections of about 15 μ thick.

3. Rapid washing in distilled water.

4. Immersion for five minutes or longer in a solution consisting of strong ammonia 10 drops, glycerine 4 c.c., distilled water 16 c.c.

5. Without washing, immerse in a 2 per cent. solution of silver nitrate, for about one minute, moving the section meanwhile.

6. Without washing, place in a 1-2 per cent. solution of formol for about 3-5 minutes.

7. After rapid washing, fix in an alcoholic solution of sodium hyposulphite.

8. Dehydrate in 95 per cent. alcohol, phenol-xylol, balsam.

The addition of 2-3 drops of gum arabic (5 per cent.) either to the nitrate bath or the reducing solution (for every 15 c.c. of the solution) improves the picture. J. V.

[130] **Method of demonstration of *Spirochæta pallida* in single microscopic sections.**—R. R. DIETERLE. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 73.

THE author has evolved a method of staining *spirochæta pallida* in sections, which on account of its reliability, simplicity and time-saving features will be welcomed by laboratory workers.

1. After fixation and cutting, place the preparations in a 1 per cent. solution of uranium nitrate in 70 per cent. alcohol at 55° C. for one half hour.
2. Wash for a moment in distilled water.
3. Pass the sections through 96 per cent. alcohol.
4. Handling the sections individually, place them in an absolute-alcoholic solution of gum mastic (10 per cent.) long enough to allow thorough infiltration—about thirty seconds. Immerse sections for an instant in 96 per cent. alcohol.
5. Transfer them to distilled water.
6. Silver for from one to six hours at 55° C. in 1 per cent. aqueous silver nitrate solution, carrying out this procedure in the dark.
7. Wash for a moment in water.
8. Develop in the following reducing solution for from five to fifteen minutes :—

Hydrochinone	1·5 gm.
Sodium sulphite	0·25 gm.
Neutral formaldehyde	10·0 c.c.
Acetone	10·0 c.c.
Pyridine	10·0 c.c.
Water, to make	90·0 c.c.

Mix and dissolve these and then add 10 c.c. of absolute-alcoholic mastic solution to make the mixture milky.

9. Wash for a moment in distilled water.
10. Dissolve out the mastic and dehydrate by transferring the sections to 96 per cent. alcohol, and then to acetone. Clear in xylol and mount in canada balsam.

The distilled water must be free from chlorides and all glassware must be likewise clean. Frozen and celloidin sections are handled with glass hooks and cover glasses with ordinary forceps. The method is also applicable to paraffin sections. R. M. S.

[131] **The Cajal and Hortega glia staining methods.**—J. H. GLOBUS. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 263.

SECTIONS obtained from brains or spinal cords which have been kept for months or years in formalin can be successfully stained by any of the methods based on the Cajal gold chloride-sublimate principle after a preliminary saturation of the tissue with ammonium bromide.

The technique is as follows:—

1. Prepare frozen sections of formaldehyde-fixed material at a thickness of from 15 to 30 microns.

2. Wash quickly in several changes of distilled water.

3. Place in a ten per cent. solution of strong ammonia water for twenty-four hours at room temperature, or for shorter periods in an incubator.

4. Carry rapidly through two changes of distilled water.

5. Place in a ten per cent. solution of pure hydrobromic acid, and let it remain there for from two to four hours.

6. Wash quickly in two changes of distilled water, to which a few drops of ammonia water is added.

7. Treat in accordance with the method selected. If it should be necessary to delay the final staining for some time, sections may be put into a 2 per cent. aqueous solution of ammonium bromide to which a few drops of neutralised formaldehyde have been added. R. M. S.

[132] **The xanthoprotein and indican content of the blood serum and cerebro-spinal fluid, with special reference to renal diseases and meningitis** (Der Xanthoprotein und Indicangehalt an Serum and Liquor cerebro-spinalis, unter besonderer Berücksichtigung von Nierenerkrankungen und Meningitis).—*Münch. med. Woch.*, 1927, lxxiv, 448.

THE xanthoprotein and indican content was estimated in 230 sera and 270 spinal fluids, and in some cases the results obtained were correlated with the amount of other nitrogen-containing substances present.

The normal xanthoprotein content of the fluid was found to be less than ten units. The indican test (Hans) was normally negative in the fluid.

In meningitis, there is usually an increase of xanthoprotein in the spinal fluid, depending on increased permeability of the meninges.

In cases of contracted kidney (even without uræmic symptoms) xanthoprotein may pass in increased quantities into the fluid. With uræmia there is nearly always an increase in the amount in the fluid, but this is not the cause of the uræmic symptoms. J. P. M.

[133] **The pathology of fatal brain-swelling** (Zur Pathologie der letalen Hirnschwellung; ein Beitrag zur Kasuistik der Fernwirkung von Hirntumoren).—E. STENGEL. *Jahrb. f. Psychiat. u. Neurol.*, 1927, xlv, 187.

IN this peculiar case, that of a woman of 53, autopsy revealed a small tumour (size of a cherry) in the extreme left occipital region. Apparently a metastatic carcinoma (from the thorax), it was the solitary neoplastic lesion of the whole brain, yet it had given rise to no symptoms by itself. The clinical picture was one of right-sided cerebellar disease, with slight pyramidal symptoms on the same side. But apart from the small tumour, the whole of the left cerebral hemisphere was in a state of marked acute swelling or œdema (well seen in the

photographs accompanying the article), and so was the mesencephalon. This latter region also exhibited numerous small blood extravasations by diapedesis. Thus, as the author remarks, his case is one of pseudotumor cerebri occurring in tumor cerebri. Not one of the clinical symptoms could be attributed to the neoplasm directly, and the problem remains, to explain the development of local œdema in two specific areas. It was the œdema that had caused all the clinical symptoms. The supposition is that the swelling of the left hemisphere had produced the changes in the mesencephalon by pressure on the vessel system of the latter, and it is suggested that, as compared with other vascular areas, the mesencephalic is one that is unduly vulnerable. The swelling of the left hemisphere as a whole must somehow be related to the presence of a tumour within it, but little is stated as to the mechanism of its production.

J. V.

[134] **Pathologico-anatomical researches with reference to the basal ganglia and their connexions** (Pathologisch-anatomische Untersuchungen mit Bezug auf die sogenannten Basalganglien und ihre Adnexe).—S. Kodama. *Schweiz. Arch. f. Neurol. u. Psychiat.*, 1927, xx, 209.

For the purpose of these investigations an examination was made of some ten human cases of varied cerebral lesions, both involving and separate from the basal ganglia, and reference is made further to three experimental animals (one dog and two cats).

The author considers himself justified in stating, contrary to the views of various workers, that cortical connexions with the basal ganglia are more definite than some of his predecessors have found. He thinks that there is a connexion between the cortex and the caudate which is definite but slight (" wenn auch schwach "). In lesions of the hemispheres he considers a slight outfall of the finest myelinated fibres of the caudate occurs.

As for the putamen, in lesions of the temporal lobe and region of the insula a slight degeneration is found in its lateral division. The globus pallidus is stated to degenerate in its posterior part from lesions of the temporal lobe; cortical lesions in the parietal region are followed by a fine degeneration in the inner and outer laminæ of the globus pallidus and in its ground-substance and ganglion cells.

In regard to the corpus Luysii and substantia nigra, a cortical connexion for the former is still doubtful, and for the latter similarly. On the other hand, degeneration of the stratum intermedium is said to follow degeneration in the cerebral peduncle, which is not the case with the substantia nigra.

It is difficult to represent in illustrations the presence of fine degenerations, and those given with the paper are not particularly clear. Further, questions of interpretation of histopathological changes require to be investigated with particular care if the cases selected are such as to render the occurrence of more than single lesions possible. The experimental evidence adduced is quantitatively insufficient.

S. A. K. W.

[135] **Central changes in Recklinghausen's disease** (Zur Kenntnis der zentralen Veränderungen bei Recklinghausenscher Krankheit).—M. BIELSCHOWSKY and M. ROSE. *Jour. f. Psychol. u. Neurol.*, 1927, XXXV, 42.

CHANGES in the central nervous system in Recklinghausen's disease can be regarded as falling into two groups: (1) developmental anomalies or dysgeneses, and (2) over-production of cell-elements of glial origin. Included among the former are changes in the vicinity of the central canal, sometimes amounting to a syringomyelia, heterotopias of white in grey substance and vice versa (spinal cord, cerebellum), nests or whorls of entangled myelinated fibres in the cerebral cortex (plaques fibromyéliniques). The latter take the form of blastoma-formations, either as neuro-epithelial tumours or as neurinomata.

The possibility of the occurrence of cases in which the changes are more or less confined to the neuraxis, peripheral nerve changes being minimal or absent, has been mooted by several workers, and a case apparently belonging to such a class is here described with much detail. The patient was a girl of 22, whose clinical symptoms were those of a nearly complete transverse lesion at the level of the lower cervical enlargement, commencing with a kind of Brown-Séquard syndrome. In addition, numerous brown pigmented flecks were visible on the skin in various areas.

Pathologically, a tumour was found within the spinal cord at the level mentioned, and it turned out to be a neurinoma. While no recognisable peripheral fibroneuromata were found, microscopical examination showed the presence of collections of cells in the cutaneous pigmented areas, suggestive of early new-formations. A long account is provided of the actual histological findings, with fine plates, and a discussion of the bearing of the case on the nature of Recklinghausen's disease is given. Briefly, the spinal tumour consisted of spindle-shaped nuclei of varying size, arranged in rows in many places though by no means everywhere, and embedded in a ground-substance staining orange with van Gieson's stain. Changes in this ground-substance had led here and there to sponge-like or cystic formations; at other spots it exhibited hyaline degeneration. Nuclei and fibres in some places tended to a whorl-arrangement. Numerous branching and intertwisting nerve-fibrils were found, almost all unmyelinated. They could be demonstrated to be encytial, that is, to run in the plasma of the tumour cells. Ganglion-cells were also found, either from the anterior cells, enclosed and degenerated, or else partaking of the nature of a new-formation (ganglioneuroma). Further, the main elements in the tumour were undoubtedly of gliogenic derivation. Thus its histology was closely allied to that of the neurinoma of peripheral nerves.

S. A. K. W.

[136] **Studies of metabolism in epilepsy. II. The sugar content of the blood. III. The blood sugar curve.**—W. G. LENNOX. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 383 and 395.

UNDER the above title the author publishes two papers dealing with the blood sugar in epilepsy.

The first is concerned with a study of 509 estimations of the fasting blood sugar level in 267 cases. The Folin-Wu method for venous blood was used, and samples were collected as a rule before breakfast. The observations were made in every conceivable circumstance with relation to the seizures and included estimations made before, during and after a simulated fit in a healthy man. After a review of literature and a full discussion with special reference to any possible connection between epileptic convulsions and those of insulin hypoglycæmia, the author concludes that there is no abnormality in the fasting blood sugar level of his patients and no direct relation between the blood sugar level and the convulsive attacks. Increase in blood sugar during the attack depends, he thinks upon the available glucose in the body.

The second paper deals with the sugar tolerance curves of 140 cases. Out of this number 24 per cent. showed curves abnormally high, 6 per cent. curves abnormally low and 70 per cent. within normal limits. In several patients there was a considerable variation both in shape and in level between successive curves. Possible reasons for this are mentioned and a further paper dealing with similar variations in normal persons is promised.

The author considers that his results fail to show any abnormality of carbohydrate metabolism which actually induces seizures. Such variations as he observed are taken to be rather a reflection of an underlying " instability of physiologic function " as to whose nature he can give no clue.

C. J. C. E.

SENSORIMOTOR NEUROLOGY.

[137] **The question of the infectious origin of acute disseminated encephalomyelitis and acute multiple sclerosis** (Zur Frage der infektiösen Aetiologie der akute disseminierten Enzephalomyelitis resp. der akuten multiplen Sklerose).—PETTE. *Münch. med. Woch.*, 1927, lxxiv, 1409.

PROFESSOR PETTE at Nonne's clinic has seen during the past year a number of cases with symptoms of encephalomyelitis which are difficult to classify. The onset is acute or subacute ; the symptoms are often vague and can seldom be referred to a circumscribed focus ; there is a tendency to great recession of the clinical manifestations. Among 25 cases seen in the last two years there were only two deaths.

These two patients died in the acute stage : both clinically and pathologically the cases were very similar. In both the disease began subacutely with rapidly progressive spastic paresis in the extremities, quickly followed by cranial nerve disturbances and, in one case, bulbar symptoms : in both cases

optic neuritis developed under observation. The multiplicity and variety of the symptoms indicated a disseminated or diffuse pathological process. In the spinal fluid the albumen was slightly increased and lymphocytes were present in excess, so that the process was probably inflammatory.

Histologically both cases showed localised patches in which the myelin sheaths were degenerated, with or without involvement of the axis cylinders; these foci were plentiful throughout the whole of the central nervous system, but had a predilection for certain parts; at the foci there was an intense growth of glial elements, especially protoplasmic glia; the vessels within the foci and in their neighbourhood were greatly dilated and showed a varying amount of infiltration: there was some infiltration in the meninges also.

There can be little doubt that the other cases observed clinically are of the same pathological nature as these two.

Henneberg, Redlich and others have recorded similar cases within the last few years, and in France Dolbfus and others have described under the title neuromyelitis optica cases which seem to be of the same kind. (A case of similar nature was described in this country by Beck at the recent combined meeting of the American Neurological Association and the Neurological Section of the Royal Society of Medicine.)

Pette inclines to regard these cases as examples of a virulent type of disseminated sclerosis, the result either of an infection with an unusually virulent strain of the organism or of particularly low resistance on the part of the patient.

Arguing from the analogy of the pathological changes he suggests that the organism concerned may be similar to those of epidemic encephalitis, of poliomyelitis, variola and rabies. J. P. M.

[138] **Encephalitis periaxialis diffusa** (Beitrag zur Kenntnis der Encephalitis periaxialis diffusa).—H. KOGERER. *Jahrb. f. Psychiat. u. Neurol.*, 1927, xlv, 109.

THE case is that of a young woman of 23, who for about 20 months had suffered from progressive amaurosis, with bilateral pyramidal symptoms, increasing loss of tone in the limbs, and a condition of moria. Bilateral optic atrophy was present (? secondary), with reduction in size of the retinal vessels. The diagnosis was uncertain; a tentative suggestion was multiple cerebral tumour. Death followed some six weeks after the patient came under observation.

At autopsy the characteristic findings of encephalitis periaxialis diffusa were discovered, most pronounced in the occipital region. The following are the usual features of the disease: preservation of the general configuration of the brain; widespread nature of the process; degeneration of white substance with preservation of grey, including the arcuate fibres; demyelination of fibres with escape of axis-cylinders; increase of glia; presence of compound granular corpuscles; involvement of vessels with perivascular infiltrates. A few references are given to recent literature.

J. V.

[139] **The etiology of infantile acquired hemiplegia.**—F. R. Ford and A. J. Schaffer. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 323.

The acute hemiplegias of infancy and childhood are uncommon, and usually occur under the age of six years, either during convalescence from some specific fever or without discoverable antecedent. The onset is febrile and is attended by focal or general convulsions. Within a few hours or a day after the onset, paralysis is noted, almost invariably of hemiplegic distribution. In the next few days the child rapidly improves, but the hemiplegia is usually, although not invariably, permanent. Subsequently, the growth of the affected limbs is retarded, and various involuntary movements may appear on that side. Mental development is more or less affected and epilepsy follows in about half of all cases. After excluding those cases associated with heart disease, meningitis, tumour of the brain, birth injury and abscess, the authors were able to collect from the records of the Harriet Lane Home forty-three cases of infantile hemiplegia. In sixteen of these, the hemiplegia followed immediately after some acute infectious disease. In seven cases the onset was associated with some local infection; pyelitis, otitis media, etc. Bacteria were not demonstrated in any of these cases, and the connection between the infection and the cerebral lesion is, of course, open to doubt. Syphilis was the etiological factor in only one case. In nineteen cases the children were considered healthy by the parents when the paralysis occurred. Seven patients were subjected to operation. In six, old scars were found, and in one on whom operation was performed several weeks after the onset arterial thrombosis and cortical softening were found to exist. Two autopsies were secured. In one, an old cyst was found which had probably resulted from a hæmorrhage, and in the other, a fresh cortical softening. In all nine cases the lesions were in the cortex.

It seems legitimate to conclude that the infantile hemiplegias which are associated with the acute infectious diseases are due to vascular lesions. Those which occur early in the course of the illness are, in the majority of cases, due to invasion of the blood stream by the primary organism or by some secondary agent. The palsies that develop during convalescence are the result of the vascular lesions in the cerebral arteries. These complications are common to all severe infections and are not in any sense specific, although apparently specific changes in the nervous system may be produced by variola, vaccinia, varicella, and perhaps by measles and mumps.

Final conclusions cannot yet be reached concerning the cause of the hemiplegias that occur in apparently healthy children, and the obvious assumption of encephalitis is not supported by anatomical evidence. In the few cases in which recent lesions have been studied only vascular lesions have been found.

R. M. S.

[140] **Two cases of flaccid paralysis in cerebral hemiplegia** (Zwei Fälle von schlaffer Lähmung bei zerebraler Hemiplegie).—I. WILHEIM. *Jahrb. f. Psychiat. u. Neurol.*, 1927, xlv, 201.

THE occurrence of flaccid hemiplegia in cerebral cases is a comparative rarity but is well recognised, although its explanation is still to seek. Two highly typical instances are here recorded, but the author is not able to offer any satisfactory view of the origination of the syndrome. The paper also summarises the differential criteria of pyramidal lesions at various levels. It would appear that cortical and mesencephalic involvement of the corticospinal tract is on the whole less likely to be followed by severe spasticity than lesions at other points in its course.

J. V.

[141] **Observations on the problem of amusia** (Bemerkungen zur Amusie-Problem).—K. M. WALTHARD. *Schweiz. Arch. f. Neurol. u. Psychiat.*, 1927, xx, 295.

A USEFUL summary of knowledge in regard to one of the obscure problems of cerebral function is followed by an elaborate account of a personal case, that of a professional musician, age 48, who had a sudden ictus with loss of speech. The aphasia remained only for a few days and from it a good recovery was made, but thereafter defects in the musical capacities of the patient became obvious. These are described at length. Summarised, they may be given as follows. Two distinct disorders were present: (1) disturbance in the appreciation of tonality, (2) disturbance in the associative use of the musical faculties that remained intact. Thus, defect in spatial appreciation of sounds was noted, also in the " mobilisation " of musical memories, and in the simultaneous synthesis of the different sound-components in an orchestral performance.

The author concludes that these defects cannot be assigned to one destructive lesion only and have not precisely a localising value; he ascribes them rather to insufficiency of nerve-impulses along anatomically intact paths or to the following of circuitous routes by impulses unable to take the usual route because of disease.

S. A. K. W.

[142] **The clinical picture of cerebral angioma racemosum** (Das klinische Bild des zerebralen Angioma racemosum arteriale).—EIMER and MEHLHOSE. *Münch. med. Woch.*, 1927, lxxiv, 836.

HÆMANGIOMATA are reckoned among the benign tumours but in certain situations, especially within the skull, they may threaten life. Intracranial angioma racemosum produces the symptoms of a cerebral tumour, but certain of its symptoms are sufficiently characteristic to permit of the clinical diagnosis of the condition. Only three cases have been recorded in which the diagnosis was made during life. In the case which the authors describe in this paper operation was declined by the patient so that the diagnosis was not actually verified.

A woman, age 23, was admitted to hospital on account of paralysis of the right arm and leg. When four years old she had had pneumonia and her mother recalled that even at that age the patient had suffered from convulsions. From that time she dragged the right leg slightly and could not use the right arm as well as the left. Year by year this disability gradually increased. After a fall when the patient was 13, redness of the left eye was noticed, but may have been present earlier. Pains and 'pins and needles' occurred later in the affected limbs; and there were also headaches, giddiness and noises in the head. Recently the relatives had noticed a prominence of the left eye.

Condition on examination.—Striking visible pulsation of the carotid arteries in the neck: the left common carotid definitely widened and the superficial arteries on the left side of the head wider and pulsating more forcibly than those on the right. Loud systolic murmur audible all over the skull, especially loudly heard when the stethoscope was placed on the bulb of the left eye: intensity much reduced by compression of left carotid; sound almost abolished by compression of both carotids.

Slight exophthalmos on the left. No perceptible pulsation of the bulbus oculi. Dilated conjunctival veins at inner angle of left eye. Eye movements full, no nystagmus; pupils reacted briskly. In the left eye vessels of the disc increased to three or four times the normal calibre and extremely tortuous. When followed towards the periphery these were seen rather suddenly to revert to the normal calibre. Right fundus normal.

Heart.—A. B. in nipple line; loud systolic murmurs in the mitral and pulmonary areas.

C.N.S.—Tongue deviated to right; slight weakness of jaw movements on right side; spasticity of right arm and leg. Power much less in right hand. Very slight wasting of right side. Loss of appreciation of light touch, temperature and painful stimuli; increased tendon jerks and positive Babinski reflex on right side.

X-ray photograph showed very broad and deep vessel furrows in distribution of middle meningeal artery, especially the anterior branches.

The clinical picture in general must consist of (1) the signs and symptoms of an intracranial tumour; (2) special signs and symptoms arising from the disturbance in the blood vessels. The signs of a focal cerebral lesion usually make their appearance first. As these tumours probably have a congenital origin, it is to be expected that symptoms would appear at an early stage (they have done so in five of the 13 cases previously on record). The condition may remain latent for many years. At puberty the growth of the tumour may be accelerated; trauma may also have a precipitating effect. Epileptiform attacks have occurred in about half of the cases.

The characteristic sign is a murmur corresponding to cardiac systole, most distinct on the skull over the site of the disease. A skiagram shows greatly dilated vessel furrows. The capital arteries are always affected; they are widened and pulsate forcibly; a systolic murmur is audible over them. In

some cases anomalies in the vessels outside the skull in the affected region aid the diagnosis, and examination of the vessels of the fundus may similarly help.

The involvement of the heart in the disease is worthy of special notice and is as yet unexplained. J. P. M.

[143] **Intracranial aneurisms.**—I. J. SANDS. *Jour. Nerv. Ment. Dis.*, 1926, lxiv, 12.

A CASE is presented and the literature reviewed. The author points out that intracranial aneurisms must be thought of when there are signs of intracranial pressure. Blood-stained spinal fluid with Kernig's sign and cervical rigidity and cranial nerve lesions make the diagnosis probable, and if the symptoms are intermittent this is strong corroborative evidence. R. G. G.

[144] **Parkinsonism following carbon monoxide poisoning.**—ROY R. GRINKER. *Jour. Nerv. Ment. Dis.*, 1926, lxiv, 18.

A DESCRIPTION is given of the lesions of white matter consequent on carbon monoxide poisoning. The author considers that these are due to asphyxiation or to deoxygenation of the blood and occur chiefly in these areas which have a poor blood supply. This explains the special incidence of lesions in the anterior part of the globus pallidus. In view of the lesion of the latter, rigidity is a common symptom and the author agrees with Vogt in believing that rigidity is a pallidal symptom and that the anterior part is related to the upper limb of the opposite side of the body and the posterior part of the lower limb. Owing to widespread interference with cortical association fibres the symptoms may be very varied. R. G. G.

[145] **Action-tremor.**—H. DE JONG. *Jour. Nerv. Ment. Dis.*, 1926., lxiv, 1.

THE author has studied tremors, specially those of paralysis agitans, by means of tracings. He concludes that action-tremor is distinct from intention-tremor and that the latter does not occur in paralysis agitans. Rest tremor and action-tremor are essentially the same and are of the same nature as clonus, being release phenomena. Clonus is due to release from control of the pyramidal system while action-tremor is due to release from the extra-pyramidal system.

R. G. G.

[146] **Dysmetric ocular ataxia** (De l'ataxie dysmétrique des yeux ; rémarques sur l'ataxie des yeux dite myoclonique (opsoclonie, opsochorie)).—C. ORZECHOWSKI. *Jour. f. Psychol. u. Neurol.*, 1927, xxxv, 1.

THE study more particularly of epidemic encephalitis has shown that various involuntary movements of the eyeballs occur which are impossible of classification as forms of nystagmus. Attention is here directed to movements classifiable as ataxia, in which there is a sort of chaotic agitation of the eyes very difficult to analyse. In one case the symptoms consisted in a kind of

dysmetria ; when the patient was requested to fix his gaze suddenly on an object placed to one side, an irregular dysmetric oscillation of the globes took place before fixation was accomplished. On his looking upward or downward the same phenomenon was present. An interesting observation was made that when he was walking and turned his eyes to fix an object his legs at once began to tremble and oscillate so that he was in danger of falling. The same patient at a later stage showed another symptom ; on looking upward and downward in the vertical direction the eyes did not move in a straight line but made a series of zig-zag movements. It should be stated that the general syndrome produced by disease in this case was one of cerebellar diplegia.

In a second case, the subject being asked to follow a moving finger, whenever the latter suddenly stopped the eyes shot past the point in a dysmetric fashion and came to rest again only after one or two large oscillations. This symptom was always more pronounced on his looking to the right. No steady and regular movement of the eyeballs seemed possible, in any direction ; hence reading became a matter of great difficulty, since the patient was unable to keep to one line.

According to the author the absence of any rhythmical element definitely prevents these peculiarities being grouped as nystagiform. They consist in failure to measure amplitude of movement and are essentially ataxic or dysmetric. He notes in particular that this ataxia is not to be confused with the " incoordination " of the oculists, which has reference to binocular fusion. A further interesting case is described of ataxia confined to one eye only.

As for myoclonic ataxia or opsoclonia, it consists in continual movement of the eyeballs, which are jerkingly displaced usually in the horizontal plane, frequently occurring in a sort of cycle and ending by a sudden jerk upwards or downwards. With these ocular symptoms are commonly found general cerebellar signs. The syndrome seems to be more characteristic of non-epidemic encephalitis than of any other morbid affection. It is conceivable that the basis of the condition is an encephalitis of the dentates.

S. A. K. W.

[147] **Paralysis of associated eye movements** (Paralisis de los movimientos asociados de los ojos).—M. Balado and E. Adrogue. *Archivos Argent. de neurol.*, 1927, i, 81.

This is a clinical and pathological study of some seven personal cases of paralysis of associated ocular movements. Among others, the interesting variety of ophthalmoplegia internuclearis is described. The usefulness of the paper is increased by a valuable bibliography of over 200 references.

S. A. K. W.

[148] **Complete nuclear ophthalmoplegia and bilateral choked disc during an acute attack of encephalitis lethargica of a hæmorrhagic type : subtemporal decompression** (Ophtalmoplégie nucléaire complète avec double stase papillaire au cours d'une poussée aiguë d'encéphalomyélite léthargique de type hæmorrhagique. Decompression sous-temporale).—Van Bogaert and Van de Briel. *Jour. de neurol. et de psychiat.*, 1927, xxvii, 492.

The main elements of this remarkable case are described in the above title, but it must be mentioned in addition (1) that the acute illness in question was a second attack of encephalitis, the first having occurred two years earlier; (2) that the patient at one stage had Argyll Robertson pupils, and (3) that though vision was for a time greatly reduced, particularly in the right eye, it was eventually recovered perfectly. The authors consider that the choked disc depended partly on the increase of intracranial tension but chiefly on actual inflammation of the optic nerve. J. P. M.

[149] **The syndrome of the corpora quadrigemina** (Sindrome cuadrigemelar).—M. R. Castex and A. F. Camauer. *Rev. oto-neuro-oft. y de cirug. neurol.*, 1927, i, 121.

Four cases of lesions of the corpora quadrigemina have been studied.

1. Softening of the region of both anterior and posterior colliculi. Symptoms : achromatopsia, pupillary paralysis, with mydriasis and abolition of direct and consensual reflex to light (on one side), paralysis of upward movements of eyes.

2. Tumour of the quadrigeminal region. Symptoms (inter alia) : paralysis of associated upward movement of eyes, anisocoria, complete abolition of pupillary reflexes, hypoacousia (right more than left).

3. Glioma of the same area. Symptoms : paralysis of upward movements, convergent strabismus, mydriasis, complete abolition of all pupil reflexes, hypoacousia becoming later total anacousia.

4. No autopsy. Symptoms : paralysis of upward movements, bilateral ptosis, tonic and clonic twitches of the eyeballs, etc.

The authors refer to an analysis by Valobra of 40 cases of quadrigeminal lesions apropos of pupillary disorders, which are given as follows :—

Complete paralysis of reactions to light and accommodation, 13.

Argyll Robertson pupil, 7.

Sluggish reactions with anisocoria, 10.

Simple anisocoria, 7.

Normal pupils, 3.

S. A. K. W.

[150] **The phenomenon of Bell, and the hyperkinetic bulbo-palpebral phenomenon** (Richiami sul fenomeno di Bell, ed osservazioni sul fenomeno bulbo-palpebral ipercinetico, nelle paralisi unilaterali del muscolo frontale).—C. NEGRO. *Giorn. de R. Accad. Med. Torino*, 1927, xc, 245.

PROFESSOR NEGRO in 1895 interpreted the familiar phenomenon of Bell (rolling up of the eyeball when the effort is made to close the ocular aperture in facial paresis or paralysis) as an example of a substitutive syncinesis ; the innervating current, unable to reach the orbiculo-palpebral group, is shunted to the elevators of the eyeball, because these form a syncinesis with the other. He argued that a similar shifting of innervation in a syncinetic group might be detected in the case of paralysis of the frontalis. If in these circumstances the patient is asked to look up, the eyeball should be seen to move farther on the affected side as the innervation intended for the frontalis is shifted to another member of the group. Professor Negro maintains this is actually the case, whereas Duverger and Barré (*Revue d'oto-neuro-ocul.*, 1926) declare it is a mere illusion, due to lowering of the upper lid and drooping of the lower lid from the facial palsy. In the present communication Negro advances fresh observations to prove that his contention is correct.

S. A. K. W.

[151] **Paratrigeminal syndrome of the ocular division of the cervical sympathetic nerve : Facial hemiatrophy** (Syndrome paratrigeminal du sympathique oculaire : hemiatrophia faciale).—VAN BOGAERT and HELMSMOORTEL. *Jour. de neurol. et de psychiat.*, 1927, xxvii, 222.

THIS is the clinical description of a case the main features of which were as follows : Paralysis of the left ocular sympathetic (myosis, slight ptosis), enophthalmos, atrophy of the skin and hairs on both eyelids and on the forehead, with loss of skin-pigment in patches ; hypalgesia and hypæsthesia in the distribution of the ophthalmic division of the left trigeminal, with paroxysms of pain in this area ; the pain extended also into the distribution of the superior maxillary division. The patient was a man of 70.

The development of facial hemiatrophy in strict association with paralysis of the cervical sympathetic is of great theoretical interest in relation to the pathogenesis of facial hemiatrophy. J. P. M.

[152] **Herpes in syphilitic patients** (Le zona des syphilitiques).—DUJARDIN. *Jour. de neurol. et de psychiat.*, 1927, xxvii, 306.

IN an interesting short paper Dujardin comments on the frequency of herpes zoster in syphilitic patients and briefly discusses the manner of its origin.

The occurrence has often been attributed to the administration of arsenic therapeutically, but herpes is relatively common in untreated cases of syphilis. During the war the author found that while among the troops in general (13,000

men at le Havre) there was an average of less than one case of herpes per thousand men per annum, the rate among 1,200 syphilitics was 4 cases of herpes per thousand. In 50 cases of herpes of all kinds 60 per cent. were intercostal and 40 per cent. were on other parts; but among 34 cases of herpes in syphilitics, less than 25 per cent. were intercostal and 75 per cent. had other localisations; Schulman found the similar proportions in his 23 cases of herpes in syphilitics. Ophthalmic herpes, especially, is often associated with syphilis.

Dujardin points out that the common distribution of herpes in syphilis corresponds to the areas of the meninges which syphilis chiefly affects, viz., on the base of the skull and in the lumbar portion of the spinal theca. He considers that syphilitic neuromeningitis renders the theca and vessels more permeable and thus opens the way for infection of the nervous system by the virus of herpes. J. P. M.

[153] **On the nature of the cremasteric reflex.**—G. Aronovitch. *Jour. Nerv. Ment. Dis.*, 1926, lxiv, 235.

An investigation of the date of appearance and constancy of the cremasteric reflex as compared with the abdominal reflex in infants. The cremasteric is the older both phylogenetically and ontogenetically and this explains its persistence in pyramidal lesions when the abdominal is lost. R. G. G.

TREATMENT.

[154] **The treatment of meningococcal meningitis** (A propos du traitement de la méningite cérébro-spinale à méningocoques).—Bouche. *Jour. de neurol. et de psychiat.*, 1927, xxvii, 311.

The author strongly recommends intraventricular injection of antimeningococcal serum at an early stage. The site of the infection is in the choroid plexus and serum injected into the spinal theca or even into the cisterna magna does not find its way into the ventricles in sufficient concentration and may not gain entrance to them at all, owing to blockage of the aqueduct. The results of intraventricular injection in children have been extremely good; in adults, in the cases hitherto reported, they have not been nearly so good, but that is probably because puncture of the ventricles has not been undertaken until the patients were *in extremis* or nearly so. J. P. M.

[155] **The treatment of trigeminal and occipital neuralgias** (Le traitement des neuralgies trigeminales et occipitales rebelles).—Muskens. *Jour. de neurol. et de psychiat.*, 1927, xxvii, 451.

In this paper the indications for and the results of operation in cases of trigeminal and occipital neuralgia are described and a number of illustrative cases are reported. In the gasserian operation the author recommends crushing as well as dividing the affected division of the nerve distal to the ganglion, because this effectively prevents regeneration of the fibres.

The paper is illustrated with some excellent diagrams and there are interesting remarks on the probable nature and cause of these neuralgias. J. P. M.

[156] **Attempts to inhibit the muscular rigidity of Parkinsonism by means of curare** (Versuche die Muskelrigidität beim Parkinsonismus durch Injektion von Kurare zu beseitigen).—KAIRIUKSCHTIS and KUTORGA. *Münch. med. Woch.*, 1927, lxxiv, 537.

AN attempt was made to abolish the excessive muscle tonus of Parkinsonism by means of local injections of curare. The preparation used was 'Kuraril,' which contains 0·5 per cent. of curare. The injections produced an amelioration of about a week's duration, but caused considerable pain at the site of injection. J. P. M.

Endocrinology.

[157] **The symptomatology of hypophyseal tumours with reference to the hypophyseal cachexia of Simmonds** (Zur Symptomatologie der Hypophysentumoren mit Hinweis auf die Simmondssche hypophysäre Kachexie).—E. REDLICH. *Jahrb. f. Psychiat. u. Neurol.*, 1927, xlv, 276.

THE syndrome of Simmonds—cachexia of pituitary origin—comprises not merely an emaciation varying in degree, but also such symptoms as dry and wrinkled skin, giving rise to a senile appearance, loss of teeth without caries, loss of pubic and axillary hair; depression, muscular weakness, feeling of coldness, subnormal temperature, bradycardia, low blood pressure; metabolic disorders, impairment of sexual functions, tendency to drowsiness and sleep, are also among the features that have been reported. The improvement practically always obtained by the administration of pituitary extracts seems to point definitely to the pituitary origin of the syndrome, which nevertheless is in fact more pluriglandular than otherwise. Besides, there is good reason to believe that some at least of the symptoms are extra-hypophyseal, and attributable to dysfunction of centres behind the pituitary, in the floor and walls of the third ventricle.

A case is here described in minute detail, conforming more or less to the type originally reported by Simmonds, and ascribed to insufficiency of the anterior lobe of the organ. There is a useful bibliography.

J. V.

[158] **Some aspects of psychoendocrine-metabolic disturbances.**—EDWARD HUNTINGDON WILLIAMS. *Medical World*, 1927, xxvii, 159.

THE close association between certain psychopathies and endocrine disturbances has been observed for years. In the investigation of the relationship between the psychoses and the endocrine system, the conditions grouped under the general term "dementia præcox" naturally present themselves for critical

investigation more emphatically than any other form of the psychoses. The frequent incidence between the onset of the disease and the puberty period, the obvious vasomotor disturbances, Abderhalden's reaction, the glucose tolerance and similar symptoms, all suggest endocrine imbalance. Also, the mental symptoms in this condition are relatively uniform, the percentage of cases high in comparison to most of the other forms of psychoses. Cushing has pointed out there is a direct relationship between the activity of the anterior pituitary and the gonads ; and the lowered bodily tone and retarded vegetative activities seen so predominantly in these cases suggest thyroidal hypofunction. Langfeldt's exhaustive investigations leave little room for doubt that hebephrenic and catatonic states are caused by endocrine disturbances. Fauser found in dementia præcox, unlike the other psychoses, " a destruction of the labumen from the sex glands as well as from the cerebral gland, but not from the other glands." Ewald and Kafka look upon " the reaction of the sex-gland albumen as being specific of dementia præcox." Lundeval and other have found leucocytosis almost invariable in certain stages of the disease. A disturbance of metabolism—usually a lowered basal metabolism—has been found by many observers in a high percentage of early dementia præcox cases. In any event, in dementia præcox of the hebephrenic and catatonic type (the paranoid being ruled out) Langfeldt voices the opinion of many observers in recent years when he says that an endocrine disturbance acting upon a constitutional instability due to an unstable endocrine system is responsible for the mental symptoms and somatic signs of the disease.

In the writer's psycho-endocrine clinic he has come in contact with mental disturbances in the early stages and has focussed his attention upon the conditions that suggest possible incipient dementia præcox, since the evidence of glandular disturbance in such cases is more positive than in other psychoses. The general plan of examination followed in each case has been : usual clinical observations ; the basal metabolism ; the blood picture ; the carbohydrate and protein metabolism ; the visceral and nervous system ; the effects of endocrine therapy—various methods of administration, observation of the effects of the different preparations used, etc. This plan has, in general, been followed by reasonably good results.

C. S. R.

Psychopathology.

PSYCHOLOGY.

[159] **The mechanism of the emotions.**—Meyer Solomon. *Brit. Jour. Med. Psychol.*, 1927, vii, 313.

The author makes the following conclusions :—

(1) The brain is necessary for emotional feeling.

(2) Emotion primarily begins centrally in the brain.

(3) Somatic or extracerebral changes are secondary to or concurrent with the cerebral reaction with its accompanying mental state.

(4) Bodily expression, skeletal or vegetative, may occur alone, be dissociated from and not accompanied by the appropriate or expected mental state.

(5) There is no evidence that reverberation from the periphery, or even from the archaic portions of the nervous system, directly induces or even augments the mental state or emotional feeling.

(6) The visceral phenomena in emotion occur not independently of but always secondarily to or concurrently with the reaction in the skeletal system.

(7) Emotion in its beginning is cortical and the reaction present takes place from the cortex downward.

C. S. R.

[160] **Psychoanalysis in theory and in life.**—T. BURROW. *Jour. Nerv. Ment. Dis.*, 1926, lxiv, 209.

THE author sets forth his standpoint towards psychoanalysis. Having been converted to Freud's teaching to begin with he now finds himself in opposition. The chief point of difference is that he regards sexuality, what is normally understood by sex, as a regression and a repressed abnormality a false substitute for the primitive 'urge' towards reproduction. He suggests that the neurotic in his refusal to accept life as it is, may be nearer the line of development, as it ought to have been, than the so-called normal civilised man. He maintains that sexuality is identical with the impulse toward self-aggrandisement and that the whole of life as ordinarily accepted is a neurotic failure of adaptation. He blames the present practice of psychoanalysis for trying to force an objective theory on to the subjective process of living, and envisages a new development which shall pay more attention to the basal requirements of life as a subjective experience. R. G. G.

NEUROSES AND PSYCHONEUROSES.

[161] **The pathogenesis of writer's cramp** (Pathogénie de la crampe des écrivains : épreuve de la re-éducation).—CALLEWAERT. *Jour. de neurol. et de psychiat.*, 1927, xxvii, 371.

CALLEWAERT considers that writer's cramp results from the association of a bad technique with inability to relax the muscles properly such as is frequently met with in various kinds of neuroses. Education in the proper technique, associated with relaxation exercises, brings about a cure. The co-ordination required is one of free movements of the phalanges with movement of the forearm round the elbow as a pivot. There should be little or no movement of the wrist and the position of the wrist should be mid-way between flexion and extension ; a position of extension is particularly to be avoided as it leads to a cramped position of the fingers. In the re-education the drawing of lines

should be practised first and then by superimposing phalangeal movements on the elbow movement loops and then letters can be introduced.

Neuritis does not bring on cramp of itself but may cause a cramped habit of the fingers to become painful. In certain cases organic troubles particularly affecting the wrist may give rise to pain during writing, but this is not a true writer's cramp. J. P. M.

[162] **Adaptation difficulties in college students.**—KARL A. MENNINGER. *Mental Hygiene*, 1927, xi, 519.

THE factors in the production of maladjustment in college students are the same factors that are at work in other problems presented to psychiatric analysis. Their genesis is to be sought partly in the material, but more largely in the shaping of the material by the environment in its most plastic period, i.e., childhood. Least important, although most conspicuous, are the restrictions and stresses peculiar to college itself; but in the rigour of the requirements of that particular life-phase, plus the disturbances in biological, social, and psychological economy that are taking place at the same time, one finds the precipitants of numerous breaks and threatened breaks. These, with varying degrees of success, the counsellor in mental hygiene, psychiatrically equipped, can repair or avert. C. S. R.

PSYCHOSES.

[163] **Chronic sepsis and mental disorder.**—T. C. GRAVES. *Jour. of Ment. Sci.*, 1927, lxxiii, 563.

THE writer gives the history of a catatonic confused case, which suggested a bad prognosis but which nevertheless recovered after treatment of septic foci. He thinks that we are justified in considering psychotic heredity as being in part, if not wholly, made up of other components. He suggests the term "septic heredity" to connote the effect of a maternal toxæmia upon the child *in utero*, while "transmissible familial infection" is another component which must not be disregarded. The progressive nature of a long-standing septic process results in a progressive deterioration of mental capacity. The hallucinations in this case were referred to the more diseased part of the head (otitis media and septic nasal sinuses). Various points correlating toxæmia and certain symptoms presented are briefly discussed.

C. S. R.

[164] **Chronic sepsis as a cause of mental disorder.**—WILLIAM HUNTER. *Jour. of Ment. Sci.*, 1927, lxxiii, 549.

THE types of mental disorders more especially concerned are those that bear the titles of dementia præcox, manic-depressive insanity, paranoid conditions, psychoneurosis, and toxic insanities. These constitute the great proportion of the admissions into mental hospitals. Certain cases illustrate, according to

the author, the effect of impacted molars, and of septic teeth generally, in producing confused mental states closely resembling dementia præcox. The existence of such teeth in one out of every eight cases of dementia præcox and manic-depressive insanity emphasizes the importance of the earlier dental history of all cases of this character, especially in the young. It is suggested that this form of psychosis should be regarded as "septic psychosis." This commonest form of psychosis, produced by long-standing chronic septic infection, exists sometimes alone, but more frequently along with and complicating other forms of psychosis, intensifying the more special mental features belonging to these latter conditions. The extent to which it is present in any case can only be determined by its removal, and the best hope that any psychotic patient can have is that his condition may prove to be largely, or possibly entirely, of the nature of a septic psychosis, and therefore one which may possibly be cured by the removal of the septic foci underlying it. Nevertheless the writer points out that it must not be thought that sepsis is the cause of all forms of insanity.

C. S. R.

[165] **Delusional projection-mechanism in a case of paralysis agitans.**—I. S. Wechsler. *Internat. Jour. of Psycho-analysis*, 1927, viii, 523.

The case quoted illustrates a projection-mechanism in a patient who was suffering from an organic disease of the nervous system. It deals with a delusion of infidelity in a man otherwise mentally normal, the delusion occurring on the basis of impotence, waning or disappearing with the return of potency and recurring with successive waves of impotence which were invariably accompanied by excessive sexual desire.

The patient was a man of 35 who complained of impotence. He had been getting weak gradually for about a year and was altogether unable to perform the sexual act for six months prior to his coming to the clinic. He had always been perfectly well and there was apparently nothing in his personal, family, or marital history to throw any light on his complaint. At a neurological examination incipient paralysis agitans was diagnosed, the result in all probability of an attack of epidemic encephalitis which was passed off as influenza. After a week on hyoscin hydrobromide the patient reported that his facultas coeundi had returned. Several months later he complained that the tablets no longer had any effect and that he was impotent once more. It was found that he was rapidly developing the classical syndrome of paralysis agitans. While still bemoaning the fact of his impotence, the patient was more alarmed about his physical condition; but the wife complained that for the first time he accused her of infidelity. The disease gradually progressed but his potency fluctuated from time to time. The delusion invariably disappeared when he was potent, and as surely returned with the recurrence of his impotence. Several attempts to gain deeper insight into the mental mechanisms failed to bring unconscious homosexual trends to the surface. After two years the patient

is utterly incapacitated physically. His delusion is still present, but it is not nearly so insistent; nor is his sexual desire so great as at first. Apparently with the waning of the desire the delusion, too, is subsiding.

The case seems of importance to the author in that a well-known projection mechanism was seen to be operating in a person with specific organic disease of the brain.

C. S. R.

[166] **Abortive disseminated sclerosis with mental type of onset** (Sclérose en plaques fruste à début mentale).—R. Targowla. *L'Encéphale*, 1927, xxii, 169.

This paper gives a clinical account of a woman of 32 who shortly after a sudden loss of consciousness developed acute maniacal excitement. Her delirium was characterised by logorrhœa, fears, delusions and hallucinations. After 12 days she became calmer and then passed into a phase of depression with anxiety. She had ideas of unreality; she wept readily; cerebration was markedly retarded. A second bout of excitement followed which was in turn succeeded by depression and that in its turn by a third manic phase.

Examination of her nervous system revealed a fixed expression and a tendency towards movement *en bloc*. Speech was somewhat scanning and dysarthric and there was some nasal quality in the voice. The pupils were unequal. All the tendon jerks were brisk and the plantars flexor in type; there were no abdominal responses. Some œdema was visible in the left macular area, surrounded by a small hæmorrhage. Later, the patient developed definite nystagmus, weakness of convergence, and tremor of the tongue. There was never any intention tremor of the limbs. The Wassermann reaction was negative, and the spinal fluid normal except for some increase in the sugar content.

During the periods of excitement some evidence of increased thyroid activity could be detected in her tachycardia, vasomotor instability, and a slight degree of proptosis.

The author has chosen to regard this case as a probable example of disseminated sclerosis, in which psychotic manifestations are predominant.

M. C.

PSYCHOPATHOLOGY.

[167] **Researches on the permeability of the meninges in some mental diseases** (Untersuchungen über die Permeabilität der Meningen bei einigen Geisteskrankheiten).—F. Weil. *Schweiz. Arch. f. Neurol. u. Psychiat.*, 1927, xx, 196.

The question of what is meant precisely by permeability of the meninges is not settled. It is known that the cerebrospinal fluid normally contains only

some of the constituents of the blood from which it is derived; under pathological circumstances the fluid content may be altered, but the nature of the mechanism behind the alterations is not clearly understood. One of the newer methods (Walter) consists in administering a watery solution of sodium bromide (·06 grm. per kilo of body weight) to the subject daily for five days; on the sixth blood is withdrawn and a lumbar puncture performed. Bromide estimation in both is made (for details see the original) and the relation of the concentration in the two is taken as the quotient of permeability (P.—Q.). The greater the meningeal permeability, the smaller is the quotient; to obtain the latter, the bromide content of the blood is divided by that of the fluid. According to Walter the average is about 3, with limits in normal states from 2.90 to 3.30.

The author has applied the technique in 13 cases of general paralysis, 16 cases of epilepsy, and 28 of the schizophrenic group. He concludes that more estimations must be made before the objective value of the method can be determined. His results do not clearly suggest that alterations in permeability accompany causally the types of psychosis that have been examined. Although increased permeability is common in general paralysis it does not appear to be constant. In about one half of the cases of "genuine" epilepsy permeability is lowered, and a similar tendency is noted in some of the schizophrenic cases.

J. S. P.

[168] **The blood-grouping of mental cases** (Untersuchungen über die Blutgruppenzugehörigkeit bei Geisteskranken).—A. Pilcz. *Jahrb. f. Psychiat. u. Neurol.*, 1927, xlv, 120.

This work is based on the examination of no fewer than 1,010 mental cases of all kinds (505 men, and an equal number women). The percentage as between the usual four groups is given as follows:

I	II	III	IV
6·6	42·4	16·1	34·9

A long and interesting table is provided, showing the percentages for different types of mental affection. Of these, one or two may be selected.

	I	II	III	IV
General paralysis (226) ...	4·4	42·9	15·9	36·8
Schizophrenia (96) ...	8·3	42·7	12·5	36·5
Epileptic psychoses (46)...	4·4	41·3	21·7	32·6
Manic-depressive (61) ...	13·1	32·8	16·4	37·7
Psychopaths (99) ...	14·4	29·3	32·3	24·2

The original should be consulted for other statistics of significance. The writer furnishes a critical discussion of his data and indicates some general conclusions. Individuals endowed with a heredo-degenerative psychotic disposition belong largely to blood-groups I and III, especially the latter. This is quite different from what obtains in the case of those "predisposed"

to general paralysis and dementia præcox. On the other hand, though in less degree, it is characteristic also of arteriosclerotic and senile psychoses; the hysterical type belongs largely to groups II and IV. However far we may still be from reducing "constitution" and "predisposition" to an objective basis, the data of this paper are a step in that direction. J. S. P.

[169] **Cerebral meiopragia from early angiosclerosis without focal ischæmia: variety of presenile arteriosclerotic dementia** (La meiopragie cérébrale par angiosclérose précoce sans ischémie en foyer: forme de démence présénile artério-scléreuse).—H. CLAUSE and J. CUEL. *L'Encéphale*, 1927, xxii, 161.

THE following case of senium præcox and progressive dementia is of interest in that subsequent pathological examination showed neither the typical changes of senility nor any focal encephalomalacic manifestations.

A taxi-driver of 49 was admitted with a six months' history of enfeeblement of the mind. He was found to be correctly orientated, but there was grave memory defect. Indefinite ideas of influence were present, but no apraxia or aphasia. Physical examination revealed emotional instability and exaggerated facial movements; the limbs were hypertonic but there were no signs of pyramidal involvement. The blood pressure was 260/140, and the blood urea 40. Wassermann reaction was negative in the blood and cerebrospinal fluid. The patient was kept under observation, and careful periodic notes were taken. Physical and mental states both deteriorated. He became disorientated and grossly demented. His gait became feebler and incoordinate; at times he was deluded; he showed various types of speech defect, partaking of stereotypy at one time, and palilalia at another. He died two and a half years after admission.

At autopsy, there was found a general thickening of the meninges; the vessels were markedly sclerosed. There was no convolutional atrophy, and on section there was no gross focal lesion, except for one tiny softening in the pons. Microscopically, there was relatively little alteration in the ganglion cells, and the cortical architectony was unchanged. There was marked neuroglial satellitosis; Bielschowsky preparations revealed no changes in the neurofibrils; there were no intracellular argentophil changes and no senile plaques. Numerous small lacunes were present in the basal ganglia with calcification of the vessel walls.

The authors exclude syphilis from the diagnosis and regard the symptoms as resulting from a relative circulatory insufficiency. M.C.

[170] **Spirochætes in the brain in general paralysis.**—A. R. GRANT. *Jour. of Ment. Sci.*, 1927, lxxiii, 595.

In a series of 50 cases of general paralysis spirochætes were found in 62·5 per cent. of the brains. Spirochætes are more likely to be found in the brain of recent acute cases than in those where the disease has run a long course.

However, in four cases of juvenile general paresis (ages ranging from 19 to 26 years at death) spirochætes were found in the brain. In cases which had been treated by malaria or tryparsamide, spirochætes were found in a much smaller percentage than in those untreated, and when present were not in such large numbers. As the number of treated cases was small, this statement requires further consideration.

C. S. R.

[171] **The mental aspects of epidemic encephalitis.**—Robert Macnab Marshall. *Jour. of Ment. Sci.*, 1927, lxxiii, 589.

The mental state of the restless, naughty child is chiefly considered. Very little has been said about the maniacal character of the child's misbehaviour, although it has more affinity with that of the maniac than with that of the delinquent, hebephrenic, or moral imbecile. There is nothing underhand or cunning about these misdemeanours. A convenient season is not chosen for the misdeeds; they are carried out in the public eye. He acts on the spur of the moment and his offences are quite devoid of *malice prepense*. On the other hand, he does not show the stolidity of the hebephrenic or the moral imbecile. He is accessible to an appeal to his better self and is not insensible to correction. Everything turns on the way in which he is corrected. If his anger or resentment is aroused, threats, abuse, and violence may be manifested. Any mental state aroused is of short duration and soon gives place to another. In short there is any amount of instability, but little or no evidence of moral depravity in the misbehaviour of the restless, naughty child. The instability is not confined to any particular faculty of the mind. All the mental processes are unduly mutable. Their ideas, their moods, and the impulses of their wills are all easily induced, and failing to develop properly, are easily supplanted by others. In consequence these children become the slaves of their environment. They obey the whim of the moment. On the other hand their restlessness is far from aimless; it is really a press of occupation. They are always busy about something, and so long as their activities are confined to useful channels they work well under supervision. The "naughty, restless child" is suffering from psychomotor excitement, and it is similar to that which may occur in the course of an attack of mania of the maniac-depressive type. What the psychologist calls general intelligence is not affected by the disorder to any appreciable extent. Recognized mental tests show that the apparent precocity of these children is not accompanied by a high intelligence quotient. On the other hand, it is quite exceptional for one of them to have a quotient below 85. Nothing in the nature of a secondary dementia is ever seen, even when the disorder has lasted so long as nine years. All that may be said about them is that their mental powers do not mature.

C. S. R.

TREATMENT.

[172] **General paralysis.**—M. M. Kund G. W. Hall and F. J. Gerty. *Jour. Amer. Med. Assoc.*, 1927, xc, 1304.

The authors describe their method of treating general paralysis by injections of foreign protein. Each course consists of a series of from eighteen to twenty-three intravenous injections of foreign protein in the form of typhoid combined vaccine (administered every second or third day) sufficient in quantity to produce a chill followed by fever with a fastigium of from 103° to 104° F. The vaccine is diluted with physiological sodium chloride solution, so that each cubic centimetre of the dilution contains 200 million dead bacilli. The initial dose is fifty million, and the number of bacilli given in subsequent doses depends entirely on the individual response of the patient, and must be gauged by the height of the temperature resulting from the previous dose. A fastigium as low as 102° F. indicates that the next dose must be increased by from 25 to 100 million bacilli. In the majority of instances an increment of 100 million dead bacilli per dose is sufficient to elicit the desired effect, so that on the eighteenth treatment the patient receives an intravenous dose of 1,800 million dead bacilli. After a two months' interval a second course of injections may be instituted. Modifications of the serological tests occur in almost every patient treated in this way. The colloidal gold curve is strikingly modified so as to approach that found in cerebrospinal syphilis, or in some cases no change above 2 or 3 is apparent. The cell count is reduced, and the blood Wassermann reaction may become negative.

In the forty-nine unselected cases thus treated, twenty-one obtained remissions sufficiently good to restore them to their former social conditions. Eight patients of the forty-nine are dead, four of whom were in an advanced stage of the disease before treatment began.

Sufficient time has not yet elapsed to allow of any definite conclusions being drawn, as the authors have published their results four months after the first course of treatments.

R. M. S.

[173] **The results of inoculated malaria in general paralytics previously suffering from the disease** (Effetti della malaria inoculata sui dementi paralitici ex-malarici).—G. Mingazzini. *Schweiz. Arch. f. Neurol. u. Psychiat.*, 1927, xx, 262.

References are given to the meagre literature bearing on the effect of artificial malaria in general paralytics who have previously suffered from ordinary malaria, and some seven personal cases are described in considerable detail. They are all those of males, and adults. The longest interval between the original malaria and the development of symptoms of general paralysis was 22 years; in most of the others it was materially shorter. The clinical type manifested was variable.

As for results, in five cases no amelioration took place, either early or late; two only showed definite improvement. The conclusions would appear to be:

(1) Previous infection with malaria by ordinary ways does not prevent general paralysis from developing subsequently;

(2) Pre-existing malarial infection does not alter the latent interval between syphilitic infection and the appearance of paralytic symptoms;

(3) The same conditions which influence favourably or not the possibility of malarial cure are equally in action in ex-malarial cases.

S. A. K. W.

Reviews and Notices of Books.

Birth Injuries of the Central Nervous System. Part I: Cerebral Birth Injuries. By FRANK R. FORD. Part II: Cord Birth Injuries. By BRONSON CROTHERS and MARIAN C. PUTNAM. With 56 illustrations. London: Baillière Tindall & Cox. 1927. Pp. 164. Price 18s. net.

A MONOGRAPH of this kind is sure to prove useful to other practitioners of medicine besides the neurologist, seeing that the obstetrician encounters cases of birth injury at the outset, while the pediatrician and the orthopædist meet them at later stages. It is paradoxically unfortunate that the subject is one which interests these various specialists, inasmuch as it may appear rather to fall between two stools (more, indeed). There can be little question that the onus in this respect lies with the obstetrician, for if anything is to be done when birth injuries declare themselves it is best to have it done quickly. Thus in not a few instances the problem presents also a surgical aspect.

One of the features of interest in the book is the attention it gives to the variety of the factors producing or responsible for what are usually termed birth "injuries"; as has long been recognised by those who are familiar with the subject, actual injury is but one of several factors, among which antenatal infection is without doubt prominent. Notwithstanding the care devoted to this part of the book, it may be questioned whether a satisfactory exposition of the phenomena has been given. Diplegia is on the face of it a semiological term; it means simply double "plegia"—double or bilateral (usually spastic) paralysis is what the clinician takes it to signify. Thus there can be no disease or nosological entity such as diplegia, convenient though the expression is. If we mistake not, however, the author of this section would prefer not to make diplegia stand for all bilateral paralyses of cerebral origin; he says, "we must admit that it is possible for symmetrically placed lesions over the convexity near the midline to simulate the clinical picture of cerebral diplegia exactly." In our view these do not *simulate* the diplegic picture; they *form* it. They are a genuine cause of diplegia. Dr. Ford maintains that the commonest cause of diplegia as he understands the term is "atrophic lobar sclerosis," following Collier in this respect, but we find no reference to the recent cogent criticisms of the latter's opinion by B. Sachs. We may ask, indeed, what is the cause of "lobar atrophic sclerosis"? It cannot be held to constitute a unique pathological condition, being itself the result of differing morbid agents. Thus ascription of diplegia largely to it does not solve the pathological problem. The author also seeks to distinguish "true diplegia" from double hemiplegia as though this were a plausible clinical procedure; but no criterion can be applied to differentiate which will hold good for all cases.

The second part of the book contains equally interesting and possibly less well recognised facts in respect of the causation of spinal injuries at birth. There are references at the end of each section, but unfortunately no index.

S. A. K. W.

The Neurotic Personality. By R. G. Gordon, M.D., D.Sc., F.R.C.P. Ed. London: Kegan, Paul, Trench, Trubner & Co. 1927. Pp. 300. Price 10s. 6d. net.

To his volume entitled *Personality*, Dr. Gordon has written a companion one, dealing on a larger scale with the neurotic personality, and analysing along interesting lines the components in the 'make-up' of that individual or clinical type. Semiology and treatment are examined in considerable detail, and the opportunity is taken of summarising and criticising various current practices in respect of the latter, and of discussing the pathological physiology of the former.

From the nosological standpoint much space is given to anxiety states, obsessions, and hysteria, and investigation here is especially careful and often illuminating, even if open to modification in several particulars. The phenomenon of defective inhibition of the antagonists deserves more consideration as a frequent hysterical motor symptom or sign than is given it. Various trophic and vascular changes accompanying hysterical phenomena are stated to be secondary, "not themselves hysterical," and dependent simply on "immobility and disuse." To this conclusion there are obvious objections; the part played by the neurovascular system in the actual manifestation of hysterical symptoms cannot be gainsaid. The author holds that anxiety states are "the invariable accompaniment of conflict within the personality," but we may ask whether he has considered the possibility of identical syndromes arising at other levels. Is "epilepsy" of bulbar centres inconceivable? And what of repercussion from structurally diseased viscera on these bulbar centres? These conditions are clinically indistinguishable from "anxiety states" yet have a different etiology.

The book is stimulating because there is much in it that is suggestive rather than established; this accounts to some extent at least for the probability that it will teach the reader most if he preserves himself an open mind in regard to not a few interpretations and conclusions. It is well written in a lucid style and has a wide appeal.

A number of slips seem to have escaped notice. "An hysterical" this or that pulls up the reader too often, and we may remind Dr. Gordon that Dejerine spelt his name without accents and that "Korsikoff" is a misspelling. "Anorhexia" is also a slip that needs correction.

Hypnosis. By Professor Paul Schilder and Dr. Otto Kauders, Vienna. Translated by Simon Rothenberg, M.D., Attending Neurologist, Jewish Hospital, Brooklyn. Nervous and Mental Monographs Series No. 46. New York: Nervous and Mental Diseases Publishing Company, 1927. Pp. 118. Price $2.50.

The difficult and still rather obscure subject of hypnosis is being slowly invaded from the physiological and clinicopathological side, as has appeared inevitable to those whose knowledge of organic neurology has for long made them impatient with the purely psychological approach to it. Phenomena in a number of respects comparable to those of the hypnotic state are met with under disease conditions of a very different kind. Trance-like states, catalepsy, akinesia, flexibilitas cerea, and so forth, seen by the clinical neurologist in both organic and functional affections, ought to be borne in mind as symptoms allied to some of those producible in a suitable individual by the hypnotist. The examination of these along physiological lines must prove fruitful when the deductions are applied to the phenomena of hypnosis. Some attempt to procede along these lines is manifest in this monograph, but it is in no little degree vitiated by a doctrinaire attitude of mind and by a readiness to accept as established what at the most should only be regarded as suggestive, if at the same time of heuristic value. We are told, for instance, that "there is no doubt" that "the sleeping centre" is situated "in the neighbourhood of the third ventricle." No allusion is made to the fundamental researches of Pawlow, with which such a bald statement is frankly incompatible, and reliance is placed on the cases reported by Lucksch and by Pette, which in point of fact are useless from the standpoint of localisation. We note in this connexion a regrettable absence of the critical spirit.

On the psychological side full consideration is given to the phenomenology of hypnosis, with due reference to the views of Freud and others. The therapeutics of the subject are also faithfully dealt with; the method is declared to be applicable to cases of neurosis, phobia, occupation neurosis, tic, stammering, hysteria, "disturbances of potency," and nocturnal enuresis. We are told incidentally that an impotent man is "only too much inclined" to be dissuaded from attempts at sexual activity, and that he "must then be forced" to make such attempts. If "serious reasons" are present, and if "the patient is familiar with the possible consequences," extra-conjugal sexual intercourse may be recommended by the physician. It is well, we think, that the profession should realise there are evidently quarters where advice of this kind is seriously tendered.

Mind and its Disorders, a Text-Book for Students and Practitioners of Medicine. By W. H. B. Stoddart. Fifth Edition. London: H. K. Lewis and Co. 1926. Pp. 593. Price 21s. net.

Successive editions of this well-known textbook mark the author's increasing conviction of the validity and importance of psychoanalytical doctrines. In

earlier editions these doctrines made their first appearance in separate chapters which were independent of the main structure of the book, but in the later editions that structure has been more and more penetrated and suffused by psychoanalytical viewpoints. A valiant attempt has been made to harmonise this new angle of approach with the clinical and pathological material which necessarily constitutes the greater part of a textbook of psychiatry. This attempt, though it cannot be said to have achieved entire success, is certainly interesting and stimulating. The author's enthusiastic acceptance of Freud's work carries with it, however, one defect of perspective which is of considerable importance in a general text-book for students. Conceptions and theories are referred to as established which certainly have not yet achieved the position which warrants such a description, and the student may well fail to appreciate that these conceptions represent only the views of a certain school, although a very important and fertile one.

The whole book has been extensively revised, and such comparatively recent topics as the malarial treatment of general paralysis and the mental sequelæ of encephalitis lethargica have been adequately considered. Except for the one criticism mentioned Dr. Stoddart's work can be confidently recommended as one of the best textbooks in our language.

Diagnosis and Spiritual Healing. By F. G. CROOKSHANK. Psyche Miniatures, Medical Series, No. 6. London: Kegan Paul & Co., 1927. Pp. 101. Price 2s. 6d. net.

Two essays are contained in this little book which will repay the attention of a wide public, both medical and lay. The first, founded on the Bradshaw lecture for 1926, deals with rival theories of diagnosis. Dr. Crookshank gives a scholarly exposition of the contrasting views of Hippocrates and Galen. The latter, which sought for a classification of diseases, has dominated medicine for the last thousand years and culminated in the absurdity of an official nomenclature. The author welcomes the signs of a return to the naturalistic method of Hippocrates, which aimed at a diagnosis of the patient as a whole with special relation to his particular disability and the method of a relief from this. In conclusion he points out that "naming the disease" is a convenience so long as the disease is considered simply as a concept and not an entity in itself and so long as our object is to arrive at a therapy suited to the patient and not exclusively to the signs he exhibits.

In the second essay, which is founded on a contribution to a discussion on spiritual healing, he justly castigates the medical men (of whom there are too many) who have too literally followed John Hunter's dictum that doctors should not think. These are always seeking to distinguish between functional and organic disease and arrive at the inevitable conclusion that "organic disease is what we say we cure but don't, while functional disease is what the quacks cure and we wish to goodness we could." He rightly points out that

a rigid distinction is absurd and that "we must rid our minds of this shibboleth before we discuss such questions as spiritual healing." He is prepared to admit the possibility of this, given sufficient evidence, since he believes in the existence of a biological impulse towards healing as exemplified by the salamander growing a new leg. He thinks that at higher levels this power might be projected to others and is of the opinion that many "healers" both within and without the profession do seem to possess a "healing power" irrespective of the drugs or other form of therapy which they employ.

R. G. G.

Idiosyncrasies. By Sir HUMPHRY ROLLESTON, Bt., K.C.B., F.R.C.P. Psyche Miniatures: Medical Series No. 8. London: Kegan Paul and Co. 1927. Pp. 119. Price 2s. 6d. net.

THE first impression left on the mind by a perusal of Sir Humphry Rolleston's interesting little volume is to how considerable an extent medical knowledge deals with probabilities rather than certainties, and how various are the interpretations of which a given set of phenomena are capable. Defining idiosyncrasy as an abnormal reaction, a functional aberration, in an otherwise normal person, and endeavouring to separate it from allergy, anaphylaxis, diathesis, constitution, and temperament, Sir Humphry provides the reader with a series of illustrations, historical and present-day, of such abnormalities in respect of food, drugs, tobacco, and so forth; more generally, in respect of both psychological and physiological reactions. The neurologist will be interested in the remarks on migraine and epilepsy, on angioneurotic oedema and asthma; and if he is of a materialistic turn of mind will be comforted by the author's patient endeavour to seek an objective explanation for these and allied syndromes. Insistence on a psychological origin for epilepsy and migraine must fail to impress any intellectually honest observer, who if he willingly admits occasional precipitation by a psychical factor nevertheless finds in innumerable other instances that causation by biochemical and other organic agencies is incontrovertible.

Dermatological Neuroses. By W. J. O'DONOVAN, M.D., Assistant Physician, Skin Department, London Hospital, etc. Psyche Miniatures; Medical Series No. 5. London: Kegan Paul & Co. 1927. Pp. 99. Price 2s. 6d. net.

ONE need not be a professional dermatologist to appreciate the charm of this little book, which deals with the psychological factor in skin affections and incidentally probes rather deeply into what is called the "seamy side" of human nature. The skin physician has his problems no less than the neurologist and psychiatrist, problems that render his practice distinctly arduous if he fails

to recognise the various factors of a non-organic kind which originate or aggravate what are ostensibly cutaneous diseases. Much information of an out-of-the-way kind is crowded into the pages, *e.g.*, that dealing with sudden blanching of the hair, or with varieties of pruritus. Dermatitis artefacta receives due consideration. Even in respect of such apparently local manifestations as lichen planus, psoriasis, eczema and alopecia the author adduces evidence pointing to the influence of etiological elements of the neurosis series. If some of the author's views are speculative rather than well-grounded they are none the less illuminating, and in any case his awareness of the play of mind on body redounds to his credit and reveals the wide-ranging outlook without which the specialist's work is vain.

Fatalism or Freedom. By C. JUDSON HERRICK, University of Chicago. Psyche Miniatures ; General series No. 10. London : Kegan Paul and Co. 1927. Pp. 106. Price 2s. 6d. net.

PROFESSOR JUDSON HERRICK'S scholarly and philosophic essay on the ancient crux of free-will versus determinism approaches the problem from the viewpoint of the biologist. If human life is a moral event it is equally true that it is a natural event, and as such it is susceptible of naturalistic interpretations. Mystical conceptions of uncaused action are out of place in any effort at a scientific appraisal of the phenomena of human activity. In ordinary parlance the individual can " make up his mind " to do this or that ; he can call on the resources of his imagination to picture himself ten years hence in such and such a position of wealth, power, comfort, ease, fame, responsibility ; he can of choice take the first and all necessary succeeding steps to secure his aim and reach his goal ; he shapes his own character, he works out his own salvation ; yet, as Professor Herrick impressively puts it, " let us not for a moment lose sight of the fact that all of these choices which thus share in the process of fabricating my own inner nature, in shaping my character, are natural functions of my bodily organs." Natural freedom means no more than that man, with a specific organisation which has grown to be what it is by natural process, exhibits the behaviour characteristic of or typical for that organisation. Natural freedom is real, unmystical, satisfying ; it embraces the finest and the best things in man's nature ; it is poles asunder from the " fatalism " of the brute ; but if we read Professor Herrick's argument aright it is the expression of orderly, determined and determining, cause-and-effect processes within him. Paradoxical as it might appear, " control is the only possible freedom " ; a genuine indeterminism would plunge him into fatalism.

Beiträge zum Ticproblem. By Dr. J. WILDER and Dr. J. SILBERMAN, Vienna. With 2 illustrations. Berlin : S. Karger. 1927. Pp. 100. Price M8.40.

IT is a little difficult to be sure of what exactly the authors of this brochure include under the term of " tic." Those who have busied themselves with the

vexed questions of involuntary movements realise the fact that distinctions on paper are easier to draw than distinctions of a clinical kind. The classical treatise of Meige and Feindel was based on the effort to differentiate involuntary movements according to their pathogenesis, and if some of their criteria are open to objection the main contention was thoroughly sound. Now-a-days, especially on the continent, neurology seems to be so entangled in the meshes of striatal lore that ascription of tics to the corpus striatum seems inevitable. We are not, therefore, in any way surprised to be given the following "definition" on the present occasion. "Tic is first of all a hyperkinesis in the form of clonus, hypertonus, or tremor, determined by functional or organic states. The functional tic is always psychogenic—either ideogenic, corresponding to a fixed idea, or psychogenic in the restricted sense, i.e., making use of preformed reflexes, reactions or automatisms. Tics are numbered with the hyperkineses of striatal origin with the exception of chorea, athetosis, and striatal tremor. All tremors occurring in an isolated form (excluding neurasthenic and senile tremor) belong to the tics. Occupation neuroses and stammering are closely allied to the tics."

An omnibus definition of this kind is completely valueless. We may ask, what evidence is there that tics are caused by striatal disease, and we get no answer. How can anyone prove that the cortex is not implicated in "striatal tics"? Assertions that tics are produced by striatal disease are based on the crudest ideas of what cerebral localisation means. If tic is to be massed with clonus, hypertonus, and tremor—a strange assortment of conditions—then is confusion worse confounded.

The useful features of the monograph would more impress the reader were he clear in his mind as to the nature of the actual hyperkinetic states described and treated by the authors.

Die Neurosen des Kindesalters; mit besonderer Berücksichtigung von Lernschwache und Schwererziehbarkeit. By Dr. W. CIMBAL, Oberarzt of the State Hospital in Altona. With 24 illustrations. Berlin and Vienna: Urban and Schwarzenberg. 1927. Pp. 509. Price M. 15; bound, M.16.50.

IT may be surprising perhaps to those less familiar with the subject how much the study of neurosis in children has been prosecuted in recent years, seeing that a volume of over 500 pages is devoted to this alone. The writer, it is true, uses the term neurosis in a somewhat elastic sense, to include conditions of mind and body not strictly neurotic. A careful and on the whole satisfying description is furnished of nervous and mental defect arising out of endocrine abnormalities and exogenous infections, but most attention is devoted to constitutional errors, endogenous and psychogenic. In some respects the book is curiously unequal, for while there are graphic and detailed studies of various clinical types their development on known pathological bases is given less

attention. Thus, for instance, the sequelæ of epidemic encephalitis receive considerably less notice than their importance merits, while congenital word-blindness is scarcely alluded to at all. The book is strongest in the wide range of the subjects it touches, in its study of pedagogic method, and above all in the excellence of the sections concerned with treatment. It is highly informative and extremely well-written.

Les Réflexes de Posture Elémentaires. By Dr. P. DELMAS-MARSALET, Interne of the Hospitals of Bordeaux. With a preface by Professors V. Pachon and H. Verger. Paris: Masson and Co. 1927. Pp. 176. Price 16 fr.

BY elementary reflex of posture Dr. Delmas-Marsalet understands what is usually termed a shortening contraction—as an example, he has worked largely with the " paradoxical contraction " of Westphal, *i.e.*, an involuntary contraction of the tibialis anticus when its ends are passively approximated. Whether this should be regarded as a reflex of posture is open to discussion. He has studied its behaviour in a large number of cases of all kinds, and considers it an " economic mechanism " of the muscle which is of preponderating importance in such static functions as posture and attitude. According to his views it is exaggerated in Parkinsonian cases and is of diagnostic value for extrapyramidal conditions, but others who have worked at the same subject have not been able to confirm them in any completeness. He considers that a fundamental antagonism can be established between elementary reflexes of posture and the phenomena of pyramidal affections, yet here again some difference of opinion unquestionably exists. However provocative of discussion this monograph represents a careful study of an interesting phenomenon and as such deserves the attention of the neurologist.

Intracranial Tumours and Some Errors in their Diagnosis. By Sir J. PURVES-STEWART, K.C.M.G., etc. London: Humphrey Milford. Oxford Medical Publications. 1927. Pp. 206. Price 12s. 6d.

THE present study is based on 121 cases of intracranial tumour whose position and character were verified by operation or at autopsy. The anatomical distribution of these is tabulated, and after a chapter on general symptoms and signs the cases are described seriatim in chapters devoted to the various regional sites; finally errors of diagnosis are discussed.

The clinical descriptions are graphic and the book should afford an excellent easy reference for those who wish to compare the problems of their own cases with the complete series of records which are here presented.

R. G. G.

Nerve Tracts of the Brain and Cord. By WILLIAM KEILLER, F.R.C.S. Ed., Professor of Anatomy and Applied Anatomy, University of Texas. New York: The Macmillan Company. 1927. Pp. 456. Price $8.

THIS book contains more information than is conveyed by its main title; it is at once a laboratory manual for the study of the anatomy of the central nervous system and an introduction to clinical neurology, ; a section is taken up with the physiology of the systems described in the first part and a final section correlates the semiology of the better known nervous diseases with this anatomo-physiological study. The author aims at several birds, and brings them all down. He seeks to obviate the common criticism of medical studies, that clinical medicine is taught separately from its basis in structure and function. With this book in his hands the student is to be envied, for he can scarcely fail to be impressed with the way in which he is led on, at the outset, from neuro-anatomy to neurophysiology, neuropathology, and clinical neurology. This is admittedly the most scientific and the most practical way of learning all about the nervous system.

The volume is well illustrated, and if not a few of the drawings are distinctly "sketchy" they will doubtless serve their purpose.

L'État Actuel de l'Étude des Réflexes. By M. MINKOWSKI, Professor in the University of Zurich. Translated from the German by Dr. Henri Ey. Revised and completed by the Author. Paris: Masson and Co. 1927. Pp. 78. Price 12 fr.

THIS is a translation into French of a recent contribution by Professor Minkowski dealing with the whole question of reflexes, using the term in its widest sense. Under it can be included the simplest aneural response of protoplasm to change in the environment, as well as the most elaborate and complex of cortical conditioned reflexes in the high-grade animal. Indeed, the claim that instincts, so-called, represent no more than a difference in degree and not in kind from reflexes is supported by the author in an interesting discussion at the close of his essay. Taken as a whole, this is one of the best recent contributions to its subject with which we are acquainted and it is well worth careful reading.

Ueber Hysterie. By Dr. ERNST KRETSCHMER, Professor of Psychiatry and Neurology in the University of Marburg. Second edition, revised and enlarged. Leipzig: Georg Thieme. 1927. Pp. 128. Price, bound, M6.

WE have already noticed the English translation of the first edition of this fine little volume in this JOURNAL (vol. vii, p. 91). To that review it is hardly necessary now to add. The author's thesis, in a word, is to the effect that hysteria is not a clinical disease-picture but a form of reaction, and his effort is to analyse what he calls the psychophysical dynamic of the condition. It is because the endeavour is made to examine the symptomatology of hysteria in terms of psychophysical function that the book is so interesting.

An Introduction to Neurology. By C. JUDSON HERRICK, Professor of Neurology in the University of Chicago. Fourth Edition, thoroughly revised. London and Philadelphia: W. B. Saunders Company, Ltd. 1927. Pp. 406. Price 12s. 6d.

THE fourth edition of Dr. Judson Herrick's deservedly popular handbook has been necessitated by the numerous advances lately made in the field of neurology. References to important recent works are added and the chapters on the cerebellum and sympathetic nervous system have been entirely rewritten. One wishes at times that certain matters, particularly those concerned with the cerebral cortex, might be more fully dealt with, but the author has thought it wise to resist the temptation to increase the size of the book.

Zwang und Zweifel (für Ärzte und Mediziner dargestellt). By DR. WILHELM STEKEL, Vienna. Part I. Berlin and Vienna: Urban and Schwarzenberg. 1927. Pp. 633. Price M30; bound, M33.

DR. STEKEL contributes yet another large volume to his personal studies of disorders of the life of impulse and affect, dealing in this instance with abnormal impulses and abnormal doubts. The material handled in his customary way is of the most diverse description, and much of it is unusually interesting. There are chapters on criminal impulses, on repetition compulsions, on imperative ideas, onanism, and so on. In the section dealing with the mechanism of production of the symptoms he states clearly and emphatically his own conclusion, "Es gibt kein Unbewusstes," maintaining on the contrary that all psychical processes are in a sense known or conscious, or capable of being conscious. His sceptical attitude in regard to the unconscious is supported by the views of others, cited at some length. He does not believe that psychoanalysis can awaken memories which before the analysis were absolutely unconscious, and states afresh the reasons which have led him to this conclusion, supporting them by clinical instances recounted at length.

Kompendium der topischen Gehirn- und Rückenmarksdiagnostik. By ROBERT BING, Professor in the University of Basel. Seventh edition, revised and enlarged. With 114 illustrations. Berlin and Vienna: Urban and Schwarzenberg. 1927. Pp. 259. Price M10; bound, M12.40.

THE seventh edition of Bing's compendium is published. It has frequently been reviewed in our pages and by this time is well-known wherever neurology is taught. Its continuing success is the best compliment that may be paid to its merits.

What Shall the Public Schools Do for the Feeble-Minded? By GUY PRATT DAVIS, Professor of Education, State Teachers College, Indiana, Penn. Harvard University Press (London: Oxford University Press). 1927. Pp. 225. Price 15s. net.

BY the "public schools" of the title the English reader must at once realise that "state-supported" schools in the English sense are meant. The object

of the writer is to furnish an exposition of the principles and practice to be followed in educating feeble-minded pupils in a given institution; in other words, it is a manual of teaching technique, based largely on the pioneer work of Binet in France and of Fernald in Massachusetts. As such, it will be found highly useful for the teaching staff in feeble-minded communities; it is extremely detailed and replete with practical suggestions, based on wide experience.